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THE ATTEMPT AT PRECISION MEASUREMENTS OF THE EAR.*

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If a "Man from Mars" were to join us in taking a course in ophthalmology and otolaryngology, he could give us a refreshing point of view. He would certainly note that the practice of otology compares favorably with the practice of ophthalmology, except in one department — that of precision measurements. For that matter, all of us at some time have given at least a passing thought to the question — Why has ophthalmology always led otology in precision diagnosis? Surely, throughout the world, this is the most neglected part of otolaryngology.

Over the entrance to a well known institute of physics in Holland, Vern O. Knudsen† noted the inscription, "Door meten tot weten" — "Through measuring to knowing," and made the comment that, although force and rhyme are sacrificed in the translation, the meaning is evident — that measuring is the beginning of exact knowledge. It means the substitution of exact measuring for theorizing; in fact, how shall we know anything satisfactorily — body temperature, blood pressure, errors of vision, impairments of hearing — except by exact measurement?

At first thought, it might seem idle to compare the eye and the ear and the clinical tests of each. These two end-organs are so different: Whereas the eye has only one function, vision, the ear is a dual sense organ — originally proprioceptive, consisting only of what we call the vestibular end-organ,

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†Personal communication.

which later assumed an auditory annex — the exteroceptive organ of hearing. Again, the eye is affected by light waves, whereas the ear is affected only by pulsations of pressure that come through a solid, a liquid or a gas.

In clinical practice, however, there is a definite analogy between the eye and the ear. In each, we study the condition and function of a highly specialized conducting mechanism and also of a highly specialized perceptive organ consisting of the terminal filaments of a nerve; consequently, it may be worth while to compare the clinical tests of the eye and of the ear; consider what each test reveals; and note the *limitations* of each test, for the eye as well as for the ear.

In studying each organ, we use both subjective and objective tests. The subjective tests of the eye are for visual acuity, color perception, field of vision and scotoma.

The tests of visual acuity are made with Snellen's and similar charts. *Limitations:* The results may vary because of the degree of illumination of the chart, the state of dark or light adaptation of the patient, the size of the pupil and other factors. We also have limitations in our audiometric measurement of hearing acuity; however, although not directly comparable to the tests of visual acuity, carefully made tests with the audiometer are nevertheless accurate. In addition to the routine determination of the type and degree of any hearing defect, we have the satisfaction of being able to determine whether a hearing defect is functional or organic. The patient cannot deceive the audiometer. All that is necessary is to make two or more tests. The audiograms will either be the same or different; and if they differ greatly, we know that the condition is functional. Of course, the differential diagnosis between psychoneurosis and malingering is determined by other factors, especially the judgment of the examiner.

Color perception is analogous to the hearing of tones of different pitch. Color vision and pitch both depend directly upon the *frequency* of the vibrations. The Ishihara and other tests of color vision are satisfactory; so are the tests of the hearing acuity for pitch. However, the tests of color vision cannot enter the field of art decoration, or occupations that demand unusual color discrimination. Few individuals can

carry in their minds the exact shade of a color, just as one seldom finds an individual who has "absolute pitch"; however, we can determine whether an individual can discriminate between different shades of color, and also the different levels of pitch.

The field of vision is accurately tested by the perimeter and Walker's screen. There are practically no limitations to these tests, except the lack of alertness to the test or the unwillingness of the patient to co-operate. In the ear, there is no analogy to the field of vision. One might say that there are two kinds of vision — the central or "spot vision," and the peripheral or "field of vision"; but there are not two kinds of hearing.

The tests for scotoma are satisfactory — although there may be difficulty in fixation or in the ability of the patient to state just where he sees the test object and where he does not. Corresponding to scotoma, we occasionally find isolated "dips" in the tonal range suggesting discrete lesions in the cochlea; these are also well demonstrated by the audiometer, especially in those instruments that have sweep frequencies for the upper tones.

The objective tests of the eye include the ophthalmoscope, the slit-lamp, the retinoscope and other instruments with which to measure refractive errors and to detect pathologic changes. The objective studies of the ear are furnished by the vestibular tests.

Although the desired degree of magnification is not always possible with the ophthalmoscope, yet its use permits a direct inspection of the inner structures, which we no doubt will never have in the study of the ear. With the slit-lamp, a great and satisfactory magnification of the anterior segment of the eye is possible. Vibration and the inability of the patient to hold the eye still are sometimes troublesome. It is difficult to apply the slit-lamp to the examination of small children.

It might seem a far cry to compare refraction with the study of middle ear function; and yet each is a definite study of a conducting mechanism. In the eye, it is the matter of determining the ability of the cornea, the aqueous, the lens and the vitreous body to conduct and refract light waves. In

the ear, it is a matter of determining the ability of the middle ear to conduct sound waves. This can be accurately determined by the comparison of the air conduction and bone conduction throughout the entire range of hearing; and can be aided by the use of an oscillator used in conjunction with an electric otoscope. Naturally, we cannot observe the motion of the drum membrane when it is exposed to sound waves; the motion is too tiny, too rapid. The ear is wonderfully sensitive: At the frequency of 1,000 cycles, a barely audible sound is heard when there is a pressure variation of only one-billionth part of atmospheric pressure in the auditory canal — which causes the drum membrane to move one-billionth of an inch. For lower frequencies, the excursion is somewhat larger. For an ordinary sound, such as the voice or the average noise in the office, the drum membrane moves one-millionth of an inch. Such movements we are not able to see. Again, we cannot watch the drum membrane move because it moves too rapidly. The eye has detectors for only one octave, from red to violet; double the frequencies for red are sensed as violet. In the ear, there are 10 octaves, from 20 to 20,000 cycles. Our eye cannot detect such rapid movements. We can, however, observe the larger motions of the drum membrane and the hammer, and to this extent we can estimate how well this portion of the ear can hear. An oscillator causes an alternate rarefaction and condensation, and produces movements of the drum membrane of about 1/100 of an inch. Such excursions are plainly visible through the electric otoscope. As to frequency, we can cause the membrane to move as slowly as three, four, five or six times per second so that we can watch the movements. Therefore, the oscillator, by reducing the frequency and increasing the amplitude, can give us an excellent concept of the performance of the drum membrane, when exposed to sound waves.

The instruments used for objective refraction are the ophthalmoscope, the retinoscope and the keratometer. The ophthalmoscope gives a fair idea within 1/3 mm. of the antero-posterior diameter of the eyeball; and a much better idea of the condition of the refracting media. Its limitations: *1.* For refraction it has the limitation of being accurate only to one or two diopters; *2.* its use is nullified for this purpose if the observer fails to control his own accommodation and is influenced by the degree to which the accommodation of

the subject is temporarily abolished by a mydriatic; *3.* it is subject to errors of judgment in determining the sharpness of focus of the picture seen; and *4.* the variation of the level of disc margins and the vessels that are easy to focus upon may be different from the macular level. The macula itself has no vessels on which to focus.

The retinoscope measures the distance from the cornea to the retina — in its various meridians — (astigmatism). Unlike the ophthalmoscope, it does not require "photographic judgment" to detect the focal sharpness upon which the ophthalmoscope depends. Even this most important single instrument for refraction must be used in conjunction with the trial case of test lenses or a phoroptometer. The accuracy of the result varies with the skill and experience of the examiner; and finally, at the trial case, rests partly upon the observation and power of sustained co-operation of the patient, who is then the observer. The condition of the refracting media of the eye examined always limits the use of the maximal resolving power of the retina. Under favorable conditions, the retinoscope is accurate to one-fourth of a diopter, as checked by the results of the trial case determinations; but it must be remembered that many a letter may be rightly named by the patient when he does not see it at all, or rightly guessed from seeing mere fragments of it.

The keratometer measures the actual curvature of the cornea in any meridian, and therefore locates the axis and the amount of astigmatism of the cornea. It does not measure an astigmatism of the retina or lens, but only of the cornea. Although variations in the curvatures of the lens are often so slight as to be negligible, occasionally they may be a definite factor in the total astigmatism.

The findings of the ophthalmoscope, the retinoscope and the keratometer are then checked with the trial case and the visual acuity range, using the Jackson crossed cylinder and astigmatic charts. Often one-eighth of a diopter of astigmatism can be detected and the axis can be found within a degree or two, provided the patient is alert and a good observer. (In a stupid patient, the results which are obtained by the use of the crossed cylinder may be misleading.) The ciliary muscle controls the focus of the conducting mechan-

ism of the eye. This muscle can be put at rest by mydriatics. The muscular control of the conducting mechanism of the ear — the tensor tympani and stapedius muscles — cannot be put at rest. As yet, we have no "mydriatics" for these muscles — nor do we need them; for there is nothing in the behavior of these muscles that is directly comparable to the act of accommodation in the eye.

As to artificial aids, in one respect that is important, hearing aids have a distinct advantage over eyeglasses. When a visual defect is the result of errors of refraction, it can be accurately measured and successfully corrected. A prescription is written for lenses which focus the image so that it will fall on the retina — for some a convex lens, and for others a concave one. The correct curvature and position of the axis can and must be provided for each eye — that is necessary, and it is also sufficient. In practice, however, no one expects eyeglasses to overcome opacities; it is universally known that vision is impossible through an opaque substance. If an eye has ulcers or scars of the cornea, opacities in the aqueous, a cataract, or an opacity of the vitreous — eyeglasses cannot overcome such conductive defects. In this respect, hearing aids have a distinct advantage over eyeglasses. If we consider the conductive lesions of the ear — atresia of the external canal; thickening or stiffening of the drum membrane; arthritis or fibrosis of the ossicular chain; or fixation of the stapes in the oval window — such lesions are analogous to the lesions of the conductive mechanism of the eye. Whereas we cannot transmit light through the dense conductive lesions of the eye by amplification of light, we actually can transmit sound through the conductive lesions of the ear, of *any type or degree*, simply by amplification of sound.

For objective evidence of the condition of the internal ear, we must rely almost entirely upon the vestibular tests. As we cannot inspect the internal ear, the problem has been to determine the condition of these parts that we cannot actually see; and in the past 25 years we have come to a far better knowledge of the condition of these hidden structures. Curiously, this first came, not in auditory, but in vestibular studies. We then attained methods of stimulating the ear and of observing objective responses, free from the fallacies introduced by the patient's subjective feelings or ideas.

In any diagnostic study of the ear, the condition of the internal ear and the VIIIth Nerve is of vital importance; and such information is far from complete without the turning and caloric tests. To be sure, we cannot learn the condition of the cochlea by making vestibular tests, or vice versa. We do not know the reason, but frequently the degree of vestibular function will prove to be greater or less than the cochlear function. Again, it is not possible to give an exact percentage of function, based on vestibular responses; we so often find that the estimated percentages of function after the turning tests are not the same as those suggested by the caloric tests. The turning tests not only give a mechanical stimulation far more vigorous than the caloric, but they probably affect the otoliths as well as the canals. Nevertheless, if we carefully note the degree of the after-turning responses of nystagmus, motion-sensing, past-pointing, pallor, sweat and nausea; and the degree of all of these same responses after the caloric tests, plus the duration of the nystagmus, based upon the total time of the douching — we can obtain far better than a general idea of the degree of function of the vestibular end-organ. In addition, we can usually differentiate between lesions of the end-organ and lesions involving the vestibular pathways in the posterior fossa; and frequently we can give valuable information in intracranial localization.

Certain caloric tests have been introduced in the attempt to simplify our old methods. The idea is to chill the ear just a trifle, and then note whether a *nystagmus* results or not. To be sure, such tests can show that the vestibular end-organ has some degree of function; but at best it limits the study to the nystagmus response alone. Frequently a patient will have a nystagmus after the average normal period of douching — 45 seconds — and yet, on prolonged douching for the total of four minutes, he may show no pallor, sweat or nausea whatever. If we rely upon the nystagmus response alone, we would consider the end-organ 100 per cent normal; but in making the complete test and studying *all* the responses, we have an entirely different idea as to the degree of function. The entire absence of constitutional responses after prolonged douching naturally demonstrates that the function is markedly subnormal. The nystagmus reflex is so sensitive that apparently it can occur when the ear transmits only a very *slight* impulse along the vestibulo-ocular tracts. Furthermore,

in cases of intracranial lesion, especially in the posterior fossa, certain of the responses may be normal, whereas other responses may be markedly subnormal or abnormal. Unless we make a complete caloric test, so that we can examine the degree of *each one* of the responses, we are shutting ourselves off from what, in many instances, would give us the most valuable information.

It has sometimes been stated that the vestibular tests are to be used only in "selected" cases — for example, in those who exhibit the *symptoms* of vertigo or staggering. The fact is, the majority of those who actually show subnormal or abnormal function, give no history at all of vestibular symptoms. It is only by making the tests that we can have the least idea of the status of the vestibular apparatus, both peripheral and central. In fact, without the vestibular tests, only one-half of the internal ear is examined; and it might well be said of us that we only half-examine the internal ear.

After the advent of the vestibular tests, the next effort toward precision was to put the auditory tests on a basis comparable to that of the vestibular tests. This became possible about 1915, by the discoveries of the vacuum tube and other modern electro-acoustic devices, which brought to us a new era in the diagnosis of hearing defects. The result is that the vestibular tests, and the developments in audiometry during the past 20 years have at last given us an approach comparable to that attained in precision measurements of the eye.

Long distance telephony, radio and talking pictures are the direct products of the creation of the vacuum tube. Audiometry is a by-product; but to the millions of hard-of-hearing persons, this by-product is far more important than the direct products.*

One promising development in audiometry has been the use of tests above the minimal threshold. We have found that some patients really hear better than their minimal threshold would indicate. After all, the important thing is to hear the *human voice*. In 1922, the writer tested patients with a simple vacuum tube amplifier; it was found that those with conduc-

*Vern O. Knudsen, Ph.D., and Isaac H. Jones, M.A., M.D.: Electro-Acoustical Instruments — Applied to the Problems of Impaired Hearing, presented before the "III. Internationale Hals., Nasen. und Ohrenarzte Kongress" in Berlin, Germany, August, 1936. Ztschr. f. Hals., Nasen. und Ohrenheilk., 40:288-294, 1937.

tive defects heard well, but that those with marked perceptive defects heard much better without the amplification. These tests of the amplified conversational voice and whisper were incorporated in the first Jones-Knudsen audiometer in 1924, which, because it contained the amplifier, was called the Audio-Amplifier. This test can be applied very simply today — using a vacuum tube *hearing aid as a diagnostic instrument.* If the deafened patient hears well with it, we know at once not only that he has a conductive defect, but that he will hear well with a hearing aid. Such a test is very helpful, in conjunction with the usual audiogram of the minimal threshold. Any simple vacuum tube amplifier is adequate — although one that furnishes high quality amplification is naturally preferable. This test can be made under noisy conditions. The audiometer, however, should of course be used in silent surroundings. Even today, tests of hearing are still being made under noisy conditions. Some audiometers are actually calibrated to be used in an "average" noise — whatever that may mean.

One of the main reasons for using a soundproof booth is that quiet surroundings are necessary in order to detect the beginning of a hearing defect. Prevention depends so much upon early detection; the patient may have already become appreciably hard-of-hearing and yet his defect will be overlooked if tests are made in an average noise.

The ideal opportunity in preventing deafness, of course, lies in the testing of school children.* The universal experience of otolaryngologists has been that patients come to them too late. There is no approach to the prevention of deafness to compare with the systematic examination of all school children. Last year in the State of California, more than $2,000,000 was spent for the blind (one-third furnished by the Federal Government, one-third by the State of California and one-third by the counties). Whether the money should come from these or other sources, the medical profession should not only bestir itself in this service but should make every effort to keep it from unworthy political influences.

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*Isaac H. Jones, M.D., and Vern O. Knudsen, Ph.D.: Audiometers and Their Place in the School Program. Jour. A. M. A., 3:8:724-725, Aug. 20, 1938.

With money, and not an excessive amount, trained workers could easily test the hearing of all school children. This would be followed by a more careful examination of those who show a hearing defect; and those who needed treatment could be sent to their own physicians. Such a simple procedure — and yet if this were done, we all realize that hundreds of thousands of children would be saved from the handicap of becoming hard-of-hearing in later life. Many such pleas have been made in different parts of the country over a period of 16 years, but in the main they have simply been voices crying in the wilderness. With the existing audiometric facilities, an ideal program would not cost much money; and the benefits to be derived from such a program would make the expenditure seem very little indeed.

As too often in the past, we will eventually be led into better ways of conducting our own work by nonmedical people.* In many branches of medicine, we have only too often followed them and not led them. If these nonmedical people give better service — although in a dangerously limited field — the patients will naturally go to them. As a profession, we neglected scientific massage; and so, due to our neglect, there developed and flourished the osteopath, the chiropractor and the host of "drugless healers." Nowadays all of us realize that fully 50 per cent of our daily work is really neuropsychiatry — regardless of our specialty; but we did not learn this soon enough. So again, to a great extent the public has come to look to the "mental healers" for the friendliness and understanding they need. Similarly, and not so very long ago, a doctor would decide, "I think I will be an eye specialist." He knew only too little of physiologic optics. Nonmedical people, not in the least trained in the diagnosis of eye conditions, *did understand physiologic optics.* Hence the optometrist, who has the good will of a large percentage of the general public. In Los Angeles, 85 per cent of the public look to the optometrist for the prescribing of glasses. How can we expect them to realize the danger of having an "eye study," limited simply to refraction?

True to form, we are again being led by nonmedical people

*Isaac H. Jones, M.D., and Vern O. Knudsen, Ph.D.: Deafness — A Social Economic and Medical Problem. Cal. and West. Med., 47:1, July, 1937.

in the careful measurements of hearing defects. The ophthalmologist of the present day can prescribe eyeglasses as well as, or better than, the optometrist; but at the moment the "audiometrist" is the one who has instruments of precision which few otolaryngologists have. Would it not be wiser for the otolaryngologist — right now — like the ophthalmologist, to be prepared to make his own precision measurements, as well as study his patient from every other angle?

It is a fact today that many otolaryngologists do not even possess a turning-chair; some at last are securing audiometers; but soundproof booths, without which any hearing test is woefully inadequate, are almost unknown in the offices of otolaryngologists. In the attempt at precision measurements, the instruments are available — and yet very few use them. It is only by early diagnosis by precision measurements that we will be able to prevent deafness in millions of people in the future. Also, by precision measurements of those already hard-of-hearing from chronic lesions, we can render the highest possible service by prescribing suitable hearing aids; however, the public will not know this and will not come to us so long as we remain unprepared to meet their need.

Recently Vern O. Knudsen and the writer presented a series of cases which might be termed, "From Wax to the Soul" — beginning with cerumen in the canal and continuing inward.* This series began with the external canal and continued inward through the various structures — in other words, from "cerumen to psychoneurosis." These cases include chronic catarrhal otitis media in which progressive deafness was stopped by tonsillectomy and resection of septum, 15 years ago; three of otosclerosis (one patient was deaf at birth, in one case deafness manifested itself after adolescence, and in the third case the deafness first appeared at the age of 47); and one case of each of the following: Arthritis of the ossicular joints, sudden deafness from acute syphilis, neuroma of the VIIIth Nerve, cerebral auditory defect and

*Isaac H. Jones, M.D., and Vern O. Knudsen, Ph.D.: What Audiometry Can Now Mean in Routine Practice. Jour. A. M. A., 3:597-605, Aug. 13, 1938.

deafness from psychoneurosis. The object of reporting this series was to put on record certain cases, the understanding of which would have been difficult without precision measurements. In fact, without precision measurements the diagnosis in many of these cases would have resolved itself into a matter of mere guesswork.

Scientifically, we should continue to note and respect every possible limitation in the available vestibular and auditory tests; but clinically, we are now perfectly able to secure a knowledge of the condition of the ear comparable to that attained in the study of the eye. If we *use* the available tests, even with the limitations they still have today, we will approach precision — but certainly not if we make vestibular tests only in "selected" cases, and hearing tests in an "average" noise.

If the day comes when each otolaryngologist will routinely use the turning-chair, the caloric test, and an audiometer in a soundproof booth, not only will he individually have the satisfaction of securing the fullest information about his patients, but the total experience will add immeasurably to our knowledge of the ear.

PRECISION MEASUREMENTS OF THE NOSE, THROAT AND EAR. A REVIEW OF LITERATURE.

NOSE AND THROAT.

Only a few years ago, Charles A. Elsberg and his associates began their novel approach toward the physics of all the special senses, by olfactory studies. It appears that this work has not yet been brought to the attention of otolaryngologists. Elsberg[1] commented that the ophthalmoscope made

it possible to study the fundus oculi; and the modern methods of perimetry enabled us to study the visual pathways — indispensable for cerebral diagnosis and localization. The vestibular tests and the finer tests of hearing have greatly advanced our understanding of cerebellar function and have become valuable aids for the recognition and localization of disease in the posterior cranial fossa. While vision and hearing are considered the higher special senses, and their investigation by more refined tests has greatly furthered diagnosis, there is every reason to believe that better methods for the study of *taste* and *smell* would yield equally gratifying results.

The olfactory receptors lie in a small area of the nasal passages, so that adequate stimuli can be brought directly to them; the olfactory bulbs are actually primary centres which lie exposed for study outside of the brain itself; the bulbs and tracts lie in a situation in which they can and must be affected by changes in intracranial pressure. Their function must be disturbed early by tumors in their neighborhood. If sufficiently sensitive tests of olfaction could be devised, slight disturbances of function of the bulbs and tracts should be recognizable.

Elsberg and Levy[2] state that the perception and identification of odors depend upon several factors: *1.* A sufficient mass of odor must come into contact; *2.* Odor must impinge with a certain degree of force; *3.* The identification of many odors depends not only upon sufficient volume and force, but also upon an effect upon the sensory receptors of the *trigeminal* nerve. There are relatively few pure olfactory stimulants. Some odors affect only the olfactory cells; many also stimulate the trigeminal nerve so that, in addition to the odor, there is a stinging, burning, cool, or hot sensation. The identification of an odor depends upon memory and association, and most odors are recognized from the combination of the olfactory and trigeminal components.

In order to study the threshold of the function of the olfactory nerve, a test should fulfill the following conditions: *1.* The test should be extremely sensitive; *2.* It should be quan-

titative, so that various degrees of alteration of the sense of smell, on one or both sides of the nose, can be recognized and measured. One must be able to speak of 20/20 smell and 10/20, 20/200 or other degrees of diminution of olfactory acuity; *3*. The test should make it possible to distinguish between the olfactory and the trigeminal factors and to study *each* of these components; *4*. The odor should be carried to the olfactory cells by some other means than that of breathing or sniffing; *5*. The volume of air and odor should be exactly controlled and measured; *6*. The force with which the air-odor mixture impinges upon the olfactory cells should be exactly and numerically measurable.

Elsberg and his associates devised a method which appears to fulfill these conditions. The odor is carried to the olfactory membrane by *direct injection* into one or both nasal passages, during a period in which the subject holds his breath. The force of the injection, therefore, takes the place of the usual nasal inspiratory movement. The volume of the injected "air-plus-odor" can be measured; and the injection is made with a known force.

The apparatus consists of test bottles, glass syringes and nosepieces. The bottle is of a definite stock size, and there is a separate bottle for each odor. The substance to be tested and which emits the odor, occupies 30 cc.; so that the space above filled with the odor is 500 cc. There is an inlet tube and an outlet tube. A known volume of air is injected with a syringe into the bottle. A pinch-cock has been closed; the bottle is "loaded" and ready for use. Nosepieces are inserted into the nostrils so that the current of odor is directed upward. The patient is instructed to hold his breath. The examiner releases the blast by a quick release of the pinch-cock on the tube to the nose.

The patient is instructed that after the blast has been released, he must remove the nosepiece and state whether there was any sensation; its character; whether an odor was perceived; whether the odor was familiar; and, finally, to name the odor. A record is made of each injection: *a*. the

volume of air injected; and *b*. the sensation experienced by the individual. This method is called the "blast injection." Successive tests are made, with increasing amounts of air injected into the bottle. The evidence that the threshold of perception is being reached is when the individual states that there is a slight odor of some kind. When a larger volume of air is injected there comes a period when the subject will state that the odor is familiar; and later, that the odor is almost identifiable. Finally, when the normal threshold for identification has been reached, the subject will name the odor.

With some substances, the trigeminal component considerably precedes the odor or olfactory component so that much larger volumes have to be injected before an odor is identified. With other substances, the trigeminal component appears only after perception or even identification of the odor. In others, the trigeminal component is so great that it is unbearable even before the olfactory component appears.

The smallest volume necessary for identification of an odor is called the "minimum identifiable odor" — the "M.I.O." — or olfactory co-efficient. This co-efficient differs for each odor and expresses the *number* of cubic centimeters of air-plus-odor required for identification of the odor. Both the volume of air-plus-odor and the pressure at which the blast is released are known quantities.

The results of studies made on normal individuals demonstrate that this new test is very sensitive.

Elsberg, Levy and Brewer, as a result of these tests, classified odors according to their exact values — as olfactory co-efficients; so that odors can now be arranged in the order of their numerical values.[3] This makes it possible to classify not only all odors that are now known, but other odors that may become known. (It is a mathematical rather than an esthetic classification.)

They also found[4] that the olfactory co-efficients vary directly as the boiling points.

In three women who happened to be tested just before the onset of menstruation, the olfactory co-efficients were found to be considerably smaller than normal — in other words a temporary olfactory *hyper*acuity.[5] A few days after the menstruation period, the coefficients were again within normal limits. An ordinary head cold may cause a great diminution of olfactory acuity — even after the period of acute congestion; in some instances several months may elapse before the olfactory coefficients return to normal. A temporary diminution may similarly be caused by drugs and articles of food. These observations demonstrate not only that the olfactory receptors are extremely sensitive, but that the blast injection test is a very sensitive method of testing even slight temporary alterations.

The same authors consider that the pressure of the blast of air-plus-odor is of greater importance for the perception and identification of the odor, than the volume.[6] The available evidence seems to indicate that both factors play a part, but that pressure is more important than volume. Most of the recent investigations have indicated that olfaction is a physical or a physico-chemical process. These studies concerning the relative importance of volume and pressure, supply evidence which points in this same direction. Even if we consider that the olfactory cells are "chemoreceptors," nevertheless olfaction appears to be basically a physical process.

In order to study the effects of odors on the trigeminal nerve-endings in the nose, and also to study olfactory fatigue, another procedure was developed — the "stream injection" test — to distinguish it from the "blast injection" tests.[7] This procedure consists of the injection into the nasal passages of a stream of air-plus-odor, for a period during which the subject is breathing through the mouth. The test bottles are the same as those used for the blast injection tests — also the nosepieces. The volume of air delivered from a tank varies between 300 and 10,000 cc. per minute. The tests are made for periods of from 15 seconds to five minutes — measured by the stopwatch. Variations in the degree of trigeminal stimulation are recorded in five stages or degrees — a code — varying from absence of sensation up to unbearable sensation. The patient also describes the character of sensation — itching, stinging, burning, warmth, coolness, pain.

They have, therefore, called those substances pure olfactory stimulants, which do not cause a trigeminal sensation, when a continuous stream of odor, at the rate of 10,000 cc. per minute, is released into both sides of the nose for several minutes. Almost all of the substances affect the trigeminus, when injected into the nose at the rate of 10,000 cc. per minute. In fact, of all the substances tested, the only ones that appear to be pure olfactory odors are coffee, musk ketone and phenyl-ethyl-alcohol. These substances are, therefore, called pure olfactory stimulants.

As to the trigeminal fatigue produced by the stream injection of odors — we do not know whether the fatigue is caused by excessive stimulation of the sensory receptors of the trigeminal nerve or whether the effect is more central. Fatigue produced by the stream injection of one odor will produce fatigue for many others, and probably for all others. In certain tests the pain is unbearable; if, by force of will, the subject continues the test, the pain soon becomes less and it often disappears entirely, even though the test is continued. Again, if stream injections for one minute are alternated with rest periods of one minute, a more or less constantly increasing fatigue is often observed. These "refractory" periods are studied and are of significance in the diagnosis of lesions that affect the trigeminal pathways.

If odors cannot be identified by sniffing, it is seldom necessary to make the blast injection tests.[8] If a patient is unable, by sniffing, to identify benzaldehyde, benzine, citral, coffee, menthol, oil of turpentine and xylol, we can conclude that he has a complete loss of smell.

For clinical tests by the blast injection and stream injection procedures, coffee, citral, oil of turpentine and benzaldehyde are recommended. In the majority of instances, coffee and citral alone will give the required information.

Elsberg's investigation[9] of the duration of olfactory fatigue — the "refractory period"— indicates that the structures concerned in fatigue of the sense of smell are in the brain itself, and in the parts of the brain that have to do with the perception and the memory of olfactory impressions. Exact areas have not yet been established, but these investigations have

already disclosed some evidence which points toward such areas.

In pressure upon the olfactory roots, the "M.I.O." is elevated; while the duration of olfactory fatigue, caused by a stream injection of odor for 30 seconds at a volume rate of 2,000 per minute, is not prolonged beyond the normal.[10] This combination of elevated M.I.O. and the normal duration of fatigue is encountered in tumors in the anterior cranial fossa underneath one or both frontal lobes of the brain. If both sides are pressed upon, the number of cubic centimeters necessary for M.I.O. is larger on both sides; but the higher elevation is found on the side on which the structures are subjected to the greater pressure. In a large series of cases, in most of the patients the olfactory tests were made and the conclusions recorded without any knowledge of the history, clinical findings or the diagnosis — except that a cerebral neoplasm was suspected. The interpretations of the olfactory tests concerned only the localization of the presumed growth. In the majority of these cases, the olfactory tests were accurate in determining whether a tumor was in the frontal lobe or under it. If the M.I.O. is high, the indication is that the tumor is under the frontal lobe. If the stream injection test demonstrates a long period of fatigue — the "refractory" period — and the M.I.O. is normal, the lesion is presumably within the frontal lobe and not under it.

In a series of 47 verified cases of tumor in or under the frontal lobe of the brain, the localization of the growth was made by the olfactory tests in 46 instances.[11] In 25 of the patients, the tumor was extracerebral and lay underneath one or both frontal lobes. In these cases, the M.I.O. was elevated on one or both sides, but the duration of fatigue was not prolonged beyond the normal. In 11 patients, the growth was within the substance of one or the other frontal lobe; in these the M.I.O. was elevated on the side of the tumor and, also, the duration of fatigue was prolonged on the same side. It is evident from these studies that the tests of olfactory acuity and olfactory fatigue, are of value for the localization of tumors in or underneath the frontal lobes of the brain.

In a further series of papers, the authors described the results obtained by quantitative olfactory tests in the localization of supratentorial tumors of the brain, especially with

reference to tumors in or under the frontal and temporal lobes. The results appear to show that this method is of value for the localization of supratentorial tumors.

The results of olfactory tests show that there is a summation of impulses in *birhinal* smell.[12] Summation of impulses occurs also, but to a lesser degree, when the olfactory receptors of one side of the nose are stimulated by an odor and those of the other side by air. There is a definite relation between the strength of the stimulus and the duration of time during which summation will occur. The study of summation of impulses from the two olfactory membranes is of value for the understanding of the physiological relation between the strength of an olfactory stimulus and the effect upon the receptor cells in the olfactory centers. It explains the cause of prolongation of fatigue on the affected side of the brain in tumors within the substance of one cerebral hemisphere.

If the olfactory membrane is stimulated by an odor at the moment when the individual is *concentrating* on the same odor, a smaller volume of odor than that normally required can be identified.[13] Under the same conditions, the odor may be recognized even during a period of complete olfactory fatigue. Olfactory fatigue appears to be the result of a temporary, partial or complete block of the pathways that connect the cerebral areas whose function is perception to those whose function is discrimination and identification of odors.

The authors give the results of tests of the olfactory and visual senses[14] which appear to indicate that both senses are governed by the same laws, and that dark adaptation in vision and so-called olfactory fatigue are basically cerebral.

An analysis of the results of tests of olfactory acuity in 40 patients with either pituitary adenoma, meningioma or aneurysm of the internal carotid artery, showed that unilateral or bilateral elevation of M.I.O., or anosmia, was found in 20 of the cases, or 72.5 per cent.[15] In seven of 23 cases of pituitary adenoma, there were no other neighborhood symptoms, and in these patients the M.I.O. was also within normal limits. There was bilateral elevation of M.I.O. in 11 of 15 patients with pituitary tumor (73 per cent); in three of seven with meningioma (43 per cent); and in four of seven with aneurysm (57 per cent). Unilateral or bilateral elevation of

M.I.O. above 30 cc., or anosmia, was found in 53 per cent of the pituitary adenomas, in 28 per cent of the meningiomas, and in 14 per cent of the aneurysms. Bilateral elevation of M.I.O. or unilateral or bilateral anosmia or elevation of M.I.O. above 30 cc. is considerably more frequent in pituitary adenoma than in meningioma or aneurysm. In the cases in which there is uncertainty, the results of tests of olfactory acuity will aid in the differential diagnosis.

The temporary lack of ability of an individual to perceive and recognize the nature of a *weak* stimulus following *strong* excitation may be called the "relative refractory state."[16] The relative refractory state is conveniently measured by its duration. The duration of the relative refractory period is, in all probability, primarily determined by the centrally situated neurones. These neurones, both from their situation and the complexity of their relationships, require a relatively long time to respond to a weak stimulus following exposure to a strong stimulus. Occlusion, facilitation, summation and distinct intensity-time effects may be demonstrated from studies of the relative refractory periods of vision and olfaction. The relative refractory periods of olfaction and of vision are frequently prolonged in the presence of supratentorial *intra*-cerebral growths. There is evidence to suggest that the duration of the relative refractory period is a measure of the number and degree of deterioration of the neurones involved. The duration of the relative refractory period in tumors of the brain is, therefore, an indication of the effects of the neoplasm on the *functions* of the affected areas.

The equations given in another paper[17] which express the relationships between the size of the pupil and the light to which the eye is exposed, are similar in the one respect that the size of the pupil appears to be related to the cube root of the intensity of the light or to some multiple of the cube root, such as the ninth root or the twenty-seventh root. It is probable, therefore, that pupillary activity is determined by the same laws as those for threshold vision and for light adaptation. Cube root relationships appear to be of basic significance for the reactions of the central nervous system to light, both for visual sensation and for the efferent impulses sent to the centers which govern the reaction of the pupil to light.

The experiences thus far gained[18] appear to indicate that:

Olfactory and visual tests are useful for the localization of supratentorial tumors of the brain and are mutually confirmatory. Tumors of the frontal lobe can be exactly localized by olfactory tests, if the tumor is so situated as to make pressure upon one or both extracerebral olfactory pathways (olfactory nerve, bulb, tract, external and internal olfactory roots). In frontal lobe tumors that lie near the superior surface of one or the other frontal lobe and are not large enough to make pressure upon the extracerebral olfactory pathways, the frontal location cannot be ascertained by the olfactory tests, but will be demonstrated by the visual tests. Slowly growing tumors in the parietal and occipital lobes may not be localized by the olfactory tests, but their location can generally be determined by visual tests. In growths in the posterior cranial fossa, the visual tests may indicate bilateral disturbance of the function of the frontal lobes if there is marked dilatation of the ventricles.

The same authors, in studies of the sense of *taste*,[19] worked out the *relation between the stimulus and the reaction time* of the individual, and reduced the calculations to formulas. They found that the equations for the sense of taste are similar to those they had previously determined for the other special senses — vision, smell and hearing; and that the same equations are applicable also to the reactions of the pupil to light.

SUMMARY: It would seem that these findings are of definite significance for a large number of vital reactions and the activity of the central nervous system.

* * *

The tests of the sense of taste have always been rather limited. Harald Tangl[20] gives a reason, in that the sense of taste is a regressive sense in man, as he tastes only with his tongue, whereas some animals, *e.g.*, fish, taste with their whole body. An infant tastes with the middle part of his tongue, but an adult determines with the sides of his tongue whether the food is pleasing or not. The whale has very few taste buds and gulps its food so quickly that it does not bother about the taste. Taste is important among horned cattle, who scrupulously select the grasses they like; the cow has about 15,000 taste buds, the antelope 50,000, and man 3,000. Man is limited to four distinct sensations from the sense of taste

— sweet, salty, bitter and sour. The sweet taste is perceived on the tongue the quickest, because the buds reacting on sweets are located at the tip of the tongue; then comes salty, and then sour taste. Bitter things are tasted at the back of the tongue. The sense of taste can be regulated. Warmth is a stimulant to the taste buds — hence warm coffee requires less sugar than cold coffee; similarly, a large amount of sugar can be put into ice cream and yet it will not seem very sweet. If the tongue is rubbed with ice, there is very little sense of taste — a good method in taking bitter medicine.

At present there is no universally accepted scale in use for describing the color of the mucous membrane, especially for the fine variations that occur in the nasal and turbinate membranes. It has been customary to use some indefinite term, such as "pale," "congested" or "dull red." In order to give a record of the gradations of color so that any reader may have a more precise concept, E. R. Hargett[21] recommends comparing any mucous membrane with the Tallquist hemoglobin scale.

EAR.

VESTIBULAR.

Frank L. Dennis[22] notes that during the past few years more vestibular studies have been made in Europe than in America. In Europe, there are many active societies, composed of otolaryngologists, neurologists and ophthalmologists, devoted directly to the study of neurologic problems in relation to eye and to ear, nose and throat studies. These groups unite in an annual congress. Valuable data on vestibular problems emanate from these meetings; and it is to be regretted that similar activities are not undertaken in our country.

Two general types of disorder affect the vestibular apparatus: peripheral lesions and central disturbances. These disorders may be either functional or organic. So the first question to be answered in any given case is — Are we dealing with a peripheral or central lesion? The next thing to be decided is whether the disorder is functional or organic. If the lesion is central and organic, the third point to be determined is —Where is it located? We try to answer these questions by neuro-otologic study — embracing the clinical history and the direct tests of the cochlear and vestibular functions.

In *peripheral* disturbances, the findings are usually alike in their essential characteristics. The spontaneous nystagmus is always horizontal-rotary, the spontaneous deviations are always bilateral and involve all segments of the body. In other words, the function as a whole is affected and the symptoms develop in a regular manner. Likewise when vestibular tests are made, the alterations involve all of the three fundamental reactions — vertigo, nystagmus and deviations. In addition, auditory defects are usually present. The character of completeness or totality is evident. Harmony is another characteristic; for instance, spontaneous deviations of the trunk and limbs are always to the same side, and the nystagmus is toward the opposite side. This same harmony is observed also in the reactions to the vestibular tests.

On the contrary, the vestibular signs in *central* lesions are characterized by extreme variability and by diverse reactions to stimulation. In contrast to those in peripheral lesions, "totality" is lacking and the characteristic findings are incompleteness and disharmony. The disturbance may be limited to one function or one reflex; the spontaneous nystagmus may be in any direction, and the deviations may be unilateral or limited to a single member or segment. The same incompleteness is observed in the reactions following the vestibular tests; one reaction may be absent, while others may be present or exaggerated; or nystagmus may not be present from the vertical canals, while good nystagmus may be obtained from the horizontal canal; or pastpointing may be in opposite directions on the two sides. Again, reactions may be dissociated; one test may reveal them and another fail to do so. Finally, auditory disturbances are usually absent.

So the two pictures are quite in contrast. In lesions of the end-organ, harmony and completeness prevail; whereas in central lesions nothing is as might be expected.

Now, as to whether a disturbance is functional or organic. In general, a peripheral, functional disturbance exhibits merely a hyper- or hypo-excitability, or, the reactions may be normal. Ordinarily in an organic, peripheral lesion, the symptoms begin suddenly with more or less intensity, and disappear after a comparatively short time. When destruction of the end-organ occurs, of course reactions are absent; in func-

tional disorders, the symptoms come and go, and exhibit less regularity and intensity.

Dennis considers it unfortunate that many otologists believe that the only usefulness of the vestibular tests is to determine merely whether the *end-organ* is active or not. He does not agree with this conception and states that many established facts indicate otherwise. He agrees with Barré[23] that there is a lamentable lack of co-operation between the three specialties; that we may be misled in trying to transfer the results of animal experimentations to man; that we have not fully realized that the whole subject is in its infancy, and that, while we have some precise data, so much remains unknown; hence not only our methods of testing are as yet imperfect, but distinct limitations are imposed on the interpretations that we can give to the findings. These facts should, however, stimulate a wider use of the vestibular tests, rather than lead to discouragement and lack of confidence in them. In fact, it is only by widespread use of the tests that we can increase our accuracy in the interpretation of the findings.

In addition to the usual vestibular tests, W. J. McNally[24] recommends that "quick tilt" tests be made. A careful analysis of the reactions of patients with internal ear or intracranial lesions is helpful in diagnosis. He considers that this test may become a routine procedure in clinical practice, just as is now possible in the laboratory.

In the examination of 30 patients with verified tumors of the VIIIth Nerve, Page Northington[25] reports that 29 showed no nystagmus, vertigo or pastpointing after the caloric test. One patient, however, showed normal responses to the turning and caloric tests, except that in douching the ear on the side of the lesion — with the head back (testing the horizontal canal) — the resulting nystagmus was oblique, instead of the normal horizontal. In addition to the good vestibular responses, there was another confusing factor — on the side of the lesion, the bone conduction was greater than the air conduction. Another patient complained of total deafness in the right ear following a head injury. The disability for totol loss of hearing in the right ear had not been allowed by the court. Cochlear tests showed the hearing to be normal in the left ear, but the patient stated he heard nothing in the right

ear. The test did not reveal malingering. The caloric reactions were normal on the left side but negative on the right. It was necessary to give an opinion, whether this patient was malingering; and if not, what was the degree of loss of hearing in the right ear. Although he claimed total deafness and the tests for malingering were not positive, the most conclusive evidence of his having unilateral total loss of hearing was the absence of any response to the caloric test. Northington states that he has never seen a patient with a traumatic ear lesion resulting in an absence of vestibular responses that had definite evidence of retaining any cochlear function. He emphasizes that this patient is one of many with a complaint of unilateral deafness, in which the important diagnostic evidence was attained by the vestibular tests. Another patient had suffered for nine months with spells of vertigo, nausea and vomiting. He was unable to carry on his work. The loss of hearing in the right ear was about 55 per cent; in the left ear, he had normal hearing. The vestibular tests gave normal responses. (After removing the adenoids and tonsils, the patient was free of dizzy spells and tinnitus, and the hearing improved to about 75 per cent in the right ear.)

Ruskin and Hyslop[26] report a case of acute syphilis of the internal ear, differentiated from trauma of the head by vestibular tests; first demonstrated by vestibular tests and later substantiated by the second stage symptoms, positive Wassermann and the demonstration of the spirocheta pallida.

There are but few tests of hearing that can be accurately applied to a child under 2 or 3 years of age, as emphasized by Max A. Goldstein.[27] The palpebral reflex — congenitally deaf children with some evidence of hearing may not show this reflex. Some, in whom no sound appreciation has ever been previously observed, do show a positive palpebral reflex. Where there can be so many exceptions to a given rule, such a test must be regarded as of questionable reliability. The vestibular tests, both turning and caloric, should be applied. Whenever a young child, profoundly deaf, is found to have normal or even subnormal vestibular responses, the assumption is justified that some degree of cochlear function is present. If the vestibular tests give no response and there is no response to hearing tests, the deafness must be regarded as profound and hopeless of acoustic stimulation. Even [illegible] is type

of patient, however, by special training in speech and lip-reading may acquire fluent, understandable speech.

The galvanic test is discussed by Shapiro[28]— although he considers it inferior to the caloric test. It was over 100 years ago that Jan Purkinje studied the subject of vertigo. He made rotation experiments upon himself and others, and noted that the resulting sensations of motion varied — with the direction of the turning and with the position of the head. He also observed the nystagmus after turning. In 1820, he constructed "a galvanic battery of 20 pairs of plates with a guitar wire leading from each pole." He put the guitar wires in both external auditory canals, and at once felt a sensation of vertigo. This was from left to right when the zinc pole was in the left ear, and in the opposite direction when the poles were reversed. After about 10 minutes he could no longer stand the intense nausea. He stated that when the current was carried through the anteroposterior diameter of the head, he did not experience any vertigo. He wrote: "It is difficult to state whether this type of vertigo arises in the brain or in the skull."

These observations of Purkinje[29] inaugurated a century of research on the phenomena of vertigo and nystagmus, including those produced by galvanic stimulation. Shapiro considers that the galvanic test possesses only a limited diagnostic value. Not only is there reason to doubt that the stimulus can be exerted only on the labyrinth itself, but as the effect is a mass reaction — *i.e.*, it is not possible to limit the stimulus to nervous elements of one canal, the clinical value of the test is still further diminished. Hans Brunner[30] notes that the galvanic test demands a meticulous technique for use in clinical examinations; but mentions a practical value of the galvanic tests introduced by his teacher, Alexander, in differential diagnosis between congenital and acquired deafness. In acquired deafness as a rule there is no response to galvanic tests, but in congenital deafness there is a definite response.

A few words may be said about vestibular chronaxia, since this is related to the subject of electric stimulation. Chronaxia may be defined as the shortest time necessary to produce a response. This subject is being widely discussed among electrophysiologists and neurologists, and has already found considerable application in certain fields of medicine.

G. Bourguignon[31] and others have experimented with chronaxia of the vestibular nerve. They used either vertigo or head movements to indicate the response. Their findings harmonize with the conclusions drawn by other observers in regard to tests of chronaxia of other mechanisms; but they found that vestibular chronaxia is the only one that varies widely in different individuals. Emotional persons show the lowest vestibular chronaxia, and the phlegmatic ones the greatest. Shapiro, therefore, considers that, in the absence of fairly definite normal standards for chronaxia of the vestibular nerve, no great practical use can be made of the galvanic test at this time.

* * *

AUDITORY.

Deafness, or even an impairment of hearing, is due almost always to a lesion of the middle ear or of the end-organ (the internal ear or VIIIth Nerve). Indeed it is a very great rarity to find that any hearing defect is traceable to a lesion of the auditory intracranial pathways, nuclei or cerebral centers. So true is this that a hearing defect always causes us to think of a lesion of the ear itself. In this matter, there is a remarkable difference between vestibular and auditory findings. Even minute lesions in the brain stem, for example, are very apt to cause subnormal or abnormal, perverted responses to the turning and caloric tests. In sharpest contrast, even gross lesions in the brain stem do not seem to have any effect whatever on the acuity of hearing. During the past 23 years the writer has observed this to be the almost invariable rule in both vestibular and auditory tests.

Confirming this experience, Crowe[32] states that a study of patients with infiltrating gliomata shows that, after the cochlear nerves enter the brain stem, their bilateral representation is so complete that the entire right temporal lobe or, indeed, the entire right cerebral hemisphere may be removed without impairing the hearing in either ear. To cause deafness, a tumor of the brain or any other type of central lesion must be extensive enough to involve the auditory pathways on both sides.

Impaired hearing for *high* tones due to conductive lesions is reported by Crowe and Guild.[33] The classical teaching,

generally accepted, has always been that all middle ear lesions cause more impairment for low tones than for high tones. In the presence of a middle ear lesion, if there is a marked impairment of hearing for high tones, and especially when there is a greater loss for the high than for the low tones — the usual diagnosis is "combined deafness"— both conductive and perceptive. The authors have long believed that this clinical picture can be produced by middle ear lesions alone. They had noted that in cases which had been diagnosed as "combined deafness," histologic studies had shown the cochlea to be normal, and the only lesions that were found in the serial sections of the temporal bone were in the *middle* ear. The only pathologic conditions observed clinically at the time of the most severe impairment of hearing were: *1.* occlusion of the pharyngeal orifices of both Eustachian tubes; and *2.* marked retraction of both drum membranes. Crowe and Guild consider that the classical idea of the effect of conductive lesions was formerly based on observations of the upper *limit* of hearing, while for the lower frequencies the *thresholds* also were determined. The development of good audiometers now makes it practicable to determine the threshold of hearing for the high as well as the low frequencies.

Previously the same authors, together with Polvogt,[34] made an extensive report of the histologic lesions in 79 ears of adults with impaired hearing for high tones, and good hearing for low tones. A study of 40 additional cases, the sections of which have been prepared since that time, confirms the previous conclusions. The typical lesion in the ears with a "gradual high tone loss" is a partial atrophy of the cochlear nerve in the osseous spiral lamina of the basal turn of the cochlea; the organ of Corti and the cells of the external sulcus are normal.

The ultimate purpose of all investigations is to learn how to prevent the development of a hearing impairment and how to restore the hearing when it is impaired. There is nothing that can be done for patients with atrophy of the organ of Corti or of the cochlear nerve. Therefore, it is of greatest importance to know that patients with more impairment for high tones than for low tones may be suffering from a middle ear rather than a cochlear lesion. Early treatment can be instituted with a good prospect for restoration of function.

The authors conclude that middle ear lesions resulting from occlusion of the pharyngeal orifice of the Eustachian tube may cause a greater impairment of hearing for high tones than for low tones; and that the judicious use of radiation therapy is worthy of an extensive clinical trial. (This treatment should be used, of course, only after a thorough adenoidectomy has failed to restore normal conditions about the tube and adequate ventilation to the middle ear.)

A continuation of this work is now reported by Crowe and Baylor.[35] Partial obstruction of the Eustachian tube in children over a long period produces retraction of the drum membranes and causes impaired hearing for *high* tones, with relatively good hearing for low tones, and sometimes a total loss of hearing by bone conduction. These unexpected and unusual findings are based on detailed observation and audiometric measurements of 60 children. In all of them, the pharyngeal orifices of the tubes were partially occluded with nodules of lymphoid tissue. (This condition in the nasopharynx is identical with granular pharyngitis but is more difficult to see.) The location of this lymphoid tissue interferes with the normal function of the tubes. They have found that the most satisfactory method of treatment is irradiation with radium or X-ray. After the hyperplastic tissue has been reduced and the tubal orifices look normal when viewed with the nasopharyngoscope, the hearing for high tones and for bone-conducted sounds often returns to the normal level, and it remains at this level as long as the Eustachian tubes are clear. After a cold, the original condition may recur, with corresponding loss of hearing. This study indicates that impairment or even total loss of hearing for high tones and for bone-conducted sounds does not necessarily imply a lesion of the cochlea. After the age of 15, secondary changes in the middle ear may become so advanced that they will not be helped by treatment. They recommend that school children in the primary grades should be examined with a nasopharyngoscope at least once a year.

It has always been the custom to test the acuity of hearing by the ability to hear the faintest audible sounds. Edmund Prince Fowler[36] emphasizes that the ear does not usually bring to consciousness such minimal audible sounds. One does not listen to them. Again, in order to test the threshold

of hearing, one must use a soundproof room or booth. There must be absolute quiet. Patients desire, above all else, to hear conversation clearly; and conversation is usually heard in fairly noisy surroundings. The environment is usually quite different from a quiet testing room. When testing at the threshold of hearing, there is a noticeable difference in the certainty of the observations. The ear with nerve deafness responds with considerable certainty. The threshold is definite; it appears suddenly from complete silence, or disappears suddenly. There is little doubt in the mind of the listener — the sound is either heard or it is not heard. On the other hand, the ear with uncomplicated conductive deafness responds with uncertainty to very faint sounds. The plotting of the threshold level may vary from moment to moment — the sound appears and disappears; it seems to have been present or have been absent for some time before the change is definitely realized. There is a lag in picking it up and in losing it. This difference in the ease of detection of threshold sound depends upon what Fowler terms the "recruitment phenomenon." It is observed in cases of perceptive defect. The recruitment phenomenon is demonstrated by throwing alternately into the two ears increasing intensities of sound. The patient compares the loudness of the sound in the two ears. The effect of tinnitus on hearing acuity may be measured by this "loudness balance" technic. Many medical and surgical procedures credited with the relief of deafness may be due to their beneficial effect on the tinnitus. The loudness of the tinnitus may be measured by matching it against the loudness of a similar frequency put into the opposite ear.

Lorente de Nó's[37] researches have suggested that, if certain neurons are missing, sufficient loudness will bring others into activity, finally reaching the point where as many nerve impulses are sent to the higher sensory stations in the brain from the diseased as from the normal ear. This would account for the finding of a "recruitment of loudness."

Fowler has also found that in some ears with nerve deafness very loud sounds appear even *louder* to a deaf than to a better hearing ear. Lorente de Nó offers an explanation as follows: Over-recruitment means that beyond a certain tone intensity, a larger number of impulses are sent to the cerebral cortex from the diseased than from the normal side. The

transmission of impulses through the primary acoustic nuclei is not continuous. It is interrupted by phases of depressed excitability. Excitation of determined channels is accompanied by inhibition of other channels, as in the spinal flexor reflex. Such inhibition in the nuclei requires accurate timing of arrival of impulses. Therefore it is thinkable that, when the VIIIth Nerve or the organ of Corti is diseased, the inhibitions in the acoustic center are less effective, and, when overloaded, allow the passage of impulses through abnormal channels, so that the cerebral cortex receives a larger number of impulses than normally. He states, "The loudness of the perceived tone must be greater, but at the same time if this explanation is true, the character of the tone must be changed and the patient cannot hear tones of very high intensity as pure tones, but rather as mixed tones, or even as noises."

Fowler states that this is exactly what he has observed clinically.

Occupational and traumatic deafness is the subject of an exhaustive study by C. C. Bunch,[28] covering the literature and his own personal work. It is an historical and also audiometric study. Industrial America is rapidly growing more and more noisy. The last 30 years have seen a marked increase in noise-producing machinery. The effect on the ear has received little attention; one only has to note the absence of studies on this subject in today's journals to convince himself that it is often unrecognized or ignored. The proof of defects produced by occupation could be determined if the hearing of employees were tested before they were employed, and at intervals during their employment. It seems improbable that such a program will be instituted even on a small scale because of the expense. As a substitute for this scientific approach, certain investigators have tested the hearing of employees who have worked in noisy surroundings, and have compared the results with those secured from tests of others working under conditions of quiet. A third method is that of investigating individual cases; this method is less accurate than either of the first two mentioned; however, regardless of the obvious sources of errors, it must be said that much of our information in all fields of medicine has been secured in this manner, and the method should not be regarded lightly. The fourth method is that of animal experimentation. This method is open to grave error; first, because

no absolutely accurate method for determining the auditory acuity of animals has been devised; second, it is quite difficult to maintain animals in health under controlled conditions for a time comparable with that under which men are working.

Bunch then gives a discussion of the hearing defects, including audiometric measurements, under many conditions — boiler makers, gunfire (from large guns and from small guns), telephone deafness, deafness in engineers, deafness in aviators, caisson deafness. The general finding is that ears that have been exposed to noise over a long period show the usual picture of cochlear defect. The deafness is most marked immediately following the period of stimulation. This is followed by a variable period of recovery. After recovery in some, the hearing loss may be so slight that it may be unnoticed by the patient and not be made evident by the hearing tests. The insidious nature of the onset of the permanent impairment of hearing produced by the excessive noise, is such that those employed under these noisy conditions are often unaware that they are becoming hard-of-hearing. These points might equally apply to any case of perceptive deafness, regardless of its etiology. The diagnosis is suggested by the history of having worked under noisy conditions. Thus it appears that occupational deafness, as such, has been inadequately described and further work should be undertaken to make possible a differential diagnosis.

The above is what Bunch found in the literature. Consequently, he made his own studies. They showed findings in agreement with the above described clinical picture. In addition, audiometric tests consistently revealed certain characteristics which conform to the diagnosis of occupational deafness.

1. An abrupt dip or gap, usually near 4,096 cycles, suggests acoustic trauma, commonly found in hunters who often are unaware of any hearing defect whatsoever.

2. With continued exposure to noise, this gap in the curve becomes deeper and broader. Artillerists, sailors who man the naval guns, and others who have been subjected to repeated explosive sounds, present this type of record.

3. Low tones, below 512 cycles, are least often involved. Boiler makers and machinists who have been in these occupa-

tions for a long time, sometimes show a loss for low tones, but the low tone loss is never so great as that for the high tones.

As to the conduction of sound waves to the cochlea — the "dual conduction theory" postulates that the low frequencies pass through the ossicular chain, while the high frequencies enter through the round window. Pohlman[39] presents objections to this theory. He also opposes the idea that the round window membrane acts as a compensation opening, essential to the reception of sound waves by the cochlea. His investigations indicate that the normal cochlea itself is relatively deaf to low tones; and that the low frequency deafness in conductive lesions is due to this fact, and not due to frequency limitations in the conduction through the middle ear.

He discusses the effects of the by-pass operation as performed by Nesfield. This operation creates a passage from the external auditory canal into the middle ear, by way of the mastoid antrum, and permits the sound pressures to reach the middle ear cavity without passing through the drum membrane. The opening of the by-pass from the external canal tends to close up, by an overgrowth which creates a diaphragm, much resembling the drum membrane. When this occurs, the hearing acuity is lowered; however, it returns to its former level on excision of the occluding membrane. Pohlman gave the acuity curves, under conditions of the by-pass open and closed, and reported an enhancement of some 15 decibels for frequencies of 1,024 and below.

He also gave results obtained in a patient who had had a radical mastoid operation. A telephone probe was applied directly to the oval window, and the sensitivity was compared with that obtained when the same probe was applied to a tooth. This is the first time that a quantitative comparison had been made between a direct and an indirect activation of the cochlea. The acuity curves were quite similar — (although an intensity increase of from 40-50 decibels was required when the contact was made with the tooth). He found it possible in this case to introduce a mechanical prosthesis, patterned after the middle ear apparatus of the bird. This prosthesis consists of a diaphragm and a rod. The diaphragm is seated in a capsule, introduced into the bony external canal; the rod imitates the columella of the bird, in that its widened foot-

plate is applied to the oval window. The transformer effect in this prosthesis consists in connecting a relatively large diaphragm, which vibrates to the sound waves in the air, with a small area applied indirectly to the perilymph. The conferred increase in hearing acuity in this case averaged 20 decibels throughout the speech range, with little effect on the low frequencies. Pohlman called attention to the probability that this prosthesis might also be employed for contact against a postoperative dehiscence in the wall of the horizontal semicircular canal; and also in those cases in which the round window area had been made accessible and protected by the method suggested by Hughson.[40]

For several years, Douglas MacFarlan[41] has emphasized the limitations of testing the hearing only at the minimal threshold, and has urged the importance of using tests with speech itself as the stimulus. Language — that coin in the realm of communication — can be only partially learned when there is poor hearing of speech; education is difficult; economically, a person is greatly handicapped; and socially, the hard-of-hearing and the deaf are often seriously maladjusted. The appreciation of these facts has led to an increasing interest in testing the hearing by actual speech itself. MacFarlan uses a phonograph with an attenuator. The index gives the percentage of correct answers. He then increases the speed of the record — which raises the pitch. The phonograph is of special help in the prescribing of a hearing aid; tests are made with and without the aid, and one can estimate the degree in the improvement in hearing contributed by the aid. In speech articulation tests made without the use of the phonograph, it is difficult for the examiner to maintain an even loudness of his voice. This fallacy is eliminated by the phonograph reproduction, in which the voice always maintains the same loudness. The crystal receiver and the crystal phonograph pickup have revolutionized the quality of speech obtainable from a record. The amplification of speech electrically and without distortion, throughout the frequency range, is now possible for the first time.

Realizing the urgent need for the standardization of audiometers, several organizations have labored on the problem of the minimal requirements for acceptable instruments. For example, the Consultants on Audiometers and Hearing Aids of the Council on Physical Therapy of the American Medical

Association have co-operated with the Sub-Committee on Audiometry and Hearing Aids of the Sectional Committee on Acoustics and Terminology of the American Standards Association. The Council on Physical Therapy[42] has compiled and published requirements for acceptable audiometers, among which are the following: Audiometers shall be equipped for testing both air and bone conduction; fixed or continuous frequencies from 128 to 8,192 cycles; the frequency of each tone shall remain within plus or minus 5 per cent of the designated value; calibration in decibels, with 5 decibels per step or less; requirements for purity of tone are described; extraneous noises shall be at least 60 decibels below the level of the test tone for frequencies of 1,024 cycles and higher frequencies, and at least 40 decibels below the level of the test tone for frequencies below 1,024 cycles; the bone conduction receiver must not produce sound in the air so that it could influence the validity of the bone conduction measurement; and the audiogram blank shall use the same base line for the results of both air conduction and bone conduction.

J. A. Keen[43] made tests by the voice, the gramophone audiometer and the pure-tone audiometer, and compared the findings by these three methods. He studied a group of school children, first with pure-tone audiogram, then the conversational voice, and then with the gramophone audiometer. He then attempted to discover some correlation between these three methods of testing. Of course, the inaccuracy of the voice test is well known. Even the best trained speaker finds it difficult to maintain the same intensity of speech; and the intensity of one person's conversational voice naturally differs from that of others. The rooms in which the voice tests are carried out have various sound reflecting powers, which depend on the dimensions of the room, the shape of the walls, the height of the ceiling and the presence or absence of curtains, carpets and large pieces of furniture. In spite of all this, he considers the voice test to be the most practical of all. No special apparatus is required and the figures obtained convey a definite idea of the hearing acuity. In the case of pupils in school, the tests of the conversational voice give a practical measure of the child's ability to hear the teacher in school.

The gramophone audiometer test resembles the voice test, in that speech is used as a basis of the test. The loudness of

the spoken numerals on the gramophone record is graduated in such a manner that each succeeding set of numerals is reproduced at an intensity of 3 decibels less than the preceding set. This is done over a range of 33 decibels, and then the voice starts again at the maximal intensity, repeating the process four times altogether, with different combinations of numbers. For those who are markedly deaf, the apparatus has a switch which causes the record to be reproduced with increased volume of sound; *i.e.*, increased loudness. Of course, it is important that each gramophone audiometer be calibrated, because there may otherwise be variations. If the decibel figures marked on the panel are accepted blindly, serious errors are bound to arise.

Pure-Tone Audiometer: When there is no speech perception, remnants of hearing power, outside the speech range of pitch and outside the loudness levels of the gramophone audiometer, can still be discovered by the pure-tone audiometer.

There are various factors which may help to explain discrepancies between the different tests. Anybody conversant with any tests of hearing knows how difficult and uncertain they are. (We cannot attain absolute precision.) The results of the gramophone audiometer were at a consistently higher level than those with voice tests. The mechanically reproduced voice is of a particularly clear type. On the whole, it appears that the gramophone test requires a smaller effort of concentration than the voice test.

The correlation between the results obtained is quite a clear one and approaches as great a degree of exactitude as one could expect, considering the widely different nature of these three methods.

An elaborate study of the interpretation in terms of decibels of the intensity of tuning forks was reported by Robert J. Hunter.[44] He concludes that the decrement of tuning forks varies so much under different conditions that a very careful technic would be necessary to get uniform results, and concludes that the audiometer is the most dependable instrument for the tests of hearing.

Aschoff and Bayer[45] note that in the last decade otologists have been concentrating on the following basic points concerning hearing tests: *1.* Continuous tone production; *2.* unvary-

ing energy of the test tones; *3.* the need of testing each ear, independent of the other; and *4.* tests of tone intensity in relation to speech intensity. They made studies of tuning forks, the Struycken monochord and the Barany noise apparatus. They discovered limitations in the use of these instruments; for example, in testing the lower tones with the tuning forks, overtones were noted. These, however, were eliminated by the application of weights, and the Bezold-Edelmann tuning forks were pronounced overtone-free. With the monochord and tuning forks, quantitative and qualitative measurements of the upper and lower limits of audition were difficult to obtain, owing to the fluctuation of the sound intensity, and the temporary lapse in the intensity of the sound waves produced. In the use of the Barany noise apparatus, the results obtained did not follow any well defined course and were subject to great variation. They consider that the value of nonelectrical instruments should not be underrated and that they will continue to have a significance in diagnostic studies; but that tuning forks and monochords do not meet the present day standards and that the audiometer affords better results because of its pure tone, volume control, and the device by which sound can be instantly produced or cut off.

Comparisons of four of the audiometers now on the market were made by Austin A. Hayden.[46] After discussing the variations in the findings with these different audiometers, he concluded that: Audiometers, despite variations, furnish the best means of testing hearing acuity; any one of the four tested will be more useful in clinical practice than any other means now available for testing and recording the hearing acuity; the lack of purity of sound wave apparently did not introduce any serious errors that were not largely explainable by other causes; a quiet room is essential; the need for a soundproof room increases as the loss of hearing to be tested decreases — that is, that one can detect a small lack of hearing only in the quiet; an audiometer should be part of the office equipment of every otologist.

BONE CONDUCTION.

Ernst Barany[47] has presented a careful study of bone conduction, which he dedicates to his father, the late Robert Barany. After a complete survey and original studies, he men-

tions three clinical situations in which a precise determination of bone conduction is most important: *1.* In suppurative lesions of the middle ear, in order to detect a beginning involvement of the internal ear; *2.* in otosclerosis, in order to determine the condition of the internal ear before a fistulating operation might be decided upon; and *3.* in cases of slight deafness, the detection of the beginning of a hearing defect — which will be of more importance in the future, when it may be possible to stop the progress of otosclerosis and lesions of the internal ear. He considers that, to give reliable information, bone conduction tests should be performed with a tuned bone conduction receiver; in a soundproof room; with a bit of rubber or wax between the teeth of the patient; with one ear excluded by masking; and with the point of application of the bone conduction receiver not indiscriminately on the mastoid process, but selected according to certain rules, which he gives in detail in this book.

Experiments made in the soundproof hearing laboratory of the University of California at Los Angeles are reported by Norman A. Watson.[48] A specially designed induction-type electrodynamic vibrator was used for all the tests. It produced vibrations over the whole audible frequency range and over a wide intensity range, with bothersome harmonics and adventitious air radiation reduced to a minimum. The vibrator was applied to the center of the forehead with a force of 370 grams weight; the vibrator button had an area of 0.665 square centimeters.

The conditions of the observer's head for perception of both pure tones and speech sounds at the lowest intensities were: mouth closed, teeth touching but not clenched, and ear canals occluded.

A tentative auditory sensation area for bone conduction was determined, on the basis of careful measurements on 18 normal hearing observers. The frequency range of the area is at least 25 to 17,000 cycles. The intensity range is at least 90 decibels at 1,000-2,000 cycles. The area is smaller than the corresponding air conduction auditory sensation area; however, it is large enough to include the frequencies and intensities of speech sounds.

To determine how well speech may be heard by bone conduction, the variation of percentage articulation, with level-

above-threshold, was studied for bone conduction, both with open and with occluded canals; and compared to that for air conduction with one ear and with two. The curves obtained in the four cases were coincident, within observational error, for a person with normal hearing. This means that for persons with normal hearing, the hearing of speech by bone conduction may attain as high a degree of perfection as that attained by the more usual method of air conduction. It also means that persons with conductive hearing impairments probably may utilize bone conduction hearing aids to as good and, in some cases, even better advantage than air conduction aids of equal fidelity.

Greenbaum, Kerridge and Ross[49] made audiometer tests in a silent room on 100 healthy young adults with normal hearing, *i.e.*, 200 normal ears. None of the individuals had ever had disease of the ear. The object was to determine what would constitute normal hearing by bone conduction. The bone conduction receiver was placed over the mastoid and the subject was asked to move the earpiece over the bone until he found the place where it sounded the loudest. Some could hear it at one point on the mastoid bone much more acutely than at other points. This optimum position has been attributed to differences in resonance of the mastoid antrum. The important consideration is that a good position should be found and kept to throughout the series of tests. In this way a most accurate idea could be obtained as to the probable success of bone conduction hearing aids. The test tones ranged from 64 to 8,192 cycles. The confusion between feeling and hearing of the low tones was easily avoided after explanation. There was no significant difference between the acuity of the right and the left ears. The results showed a somewhat wide variation, even though the tests were carefully done on intelligent and co-operating adults. It is impossible to say how much the differences in acuity were due to perception by the cochlea, and how much the differences in the condition of the skin and the mastoid bone.

E. Godfrey Burr and Hector Mortimer[50] report the technic for more precise audiometric measurements; and a soundproof chamber is described. The patient sits at a definite distance from a loud speaker. Outside of the soundproof chamber, the observer is able to see the patient through the

window. To avoid errors, the patient replies to the sounds which are automatically recorded. The particular object of this technic is to give precise measurements of changes in the loss of hearing. This type of installation is also applicable to other fields of investigation, such as comparison of the threshold with and without a hearing aid, and comparison of levels at which articulation is clear.

The degree of soundproofing — the keenest ear is unable to detect noise from the outside; two persons could not agree whether or not they heard a motor horn blown within a foot of the wall of the building; the sound of a hammer and chisel applied to the main building structure close to the soundproof booth is just audible within the chamber. It is desirable to have the chamber disconnected from the main building — although in practical uses it is easy to locate the sources of disturbance and stop them.

Yver and Barrat[51] comment on their own audiometric tests. They emphasize that those with only minor hearing defects have difficulty in deciding at which intensity they can hear, unless the test is made in a soundproof chamber; they advise that a true test be divided into two examinations, because of the fatigue of the patient; and report their audiograms in many diseases of the ear.

MASKING.

Hulka[52] emphasizes that a masking apparatus must make enough noise, but not too much noise, as it then would mask not only the better ear but the one being tested. As Harvey Fletcher[53] has shown, when a pure tone below 500 cycles acts on a drum membrane, the simple regular vibrations, provided they reach certain intensities, are transformed into complex waves. The cochlea hears the fundamental tone plus harmonics. This produces a masking effect over the range heard. The ideal is to produce a "masker" that *1.* consists of a proper mixture of pitches and intensities; and *2.* reaches only one ear, leaving the opposite ear unencumbered for delicate hearing tests.

Hulka describes the three devices for masking: *1.* The telephone buzzer; *2.* electric door buzzer; *3.* current of air blown into the meatus. The telephone buzzer does not suffi-

ciently mask the tones above 2,000 cycles, and it interferes with the hearing being tested. The door buzzer produces even more extraneous noise. Blowing of air against the drum membrane gives very little extraneous noise, is satisfactory for air conduction tests but not so satisfactory for bone conduction tests. Hulka constructed an apparatus, combining the door buzzer and an air current produced by a rubber balloon. The resultant noise resembles a deep hum, comparable to a tone of about 128 cycles; also a metallic din, with pitch between 1,000 and 2,000 cycles. There is little extraneous noise and about 60 decibels of effective complex masking tone. Audiograms are presented which describe the elimination of the bone conduction and air conduction shadow curves.

A method of determining the minimal sound intensity to be employed in auditory masking is described by Antonio Ciocco.[54] The necessity of masking is generally recognized, yet insufficient consideration has been given to the technic. The masking noise should be neither too strong nor too weak — of sufficient intensity to eliminate hearing in the masked ear, but not of sufficient intensity to interfere with the other ear that is being tested. Ciocco employs the Weber test; a tuning fork of 512 double vibrations is held in the midline of the head, and the masking noise is varied until it is just loud enough to make it inaudible in the masked ear. He also tested 500 children whose reaction to the Weber test was not lateralized, and found that the average minimal intensity for masking was 60 decibels.

Dishoeck[55] presents an instrument for testing the patulency of the Eustachian tube — the "Pneumophon." Hearing is most acute when the air pressure on both sides of the drum membrane is equal. As the air pressure in the middle ear is often negative in pathologic conditions, a quantitative estimation of the degree of the negative pressure makes possible a more exact diagnosis of the degree of tubal insufficiency. If the air pressure in the external canal is gradually lowered, a distinct zone of optimal hearing will be noted — corresponding to the balance of air pressure on both sides of the drum membrane. A small and simple apparatus is described by Dishoeck. A noise is introduced by an electrical buzzer in a small soundproof box; this noise is conducted to the external canal by a thick-walled rubber tube with an olive-shaped tip.

The rubber is connected by one tube with a water manometer and by another tube with a rubber ball. By pressing the rubber ball, the positive pressure will appear in the whole system; this can be exactly measured by the manometer. By releasing the rubber ball, which was previously pressed, the pressure will be lowered by the suction of air and the pressure will gradually change from positive to negative. By noting the appearance of the zone of optimal hearing, the moment of the establishment of the exact balance of the air pressure in the middle ear and in the external canal can be checked. In the normal individual, the zone of distinct hearing is —2 to +2. In tubal insufficiency, one notes that the best hearing occurs during a negative pressure of from —6 to —14. By insufflation of air into the middle ear, a normal air pressure in the middle ear is established in a relatively short time; but gradually the negative pressure reappears. Dishoeck considers it possible that benefit may come by insufflating various gases instead of air — because certain gases may be absorbed less rapidly than air itself.

Hughson, Ciocco and Palmer[56] mention that otologists are beginning to realize that their total contribution to the subject has been relatively insignificant — because the studies have not been applied to the patients who have not received the assistance which they desperately need. This awakened interest will not only add to our knowledge, but also to therapeutic possibilities. Early diagnosis and treatment have been regarded by too many of us as too time-consuming; and the results of therapy have been too disappointing to warrant the sustained interest of the busy practitioner. Whatever the ultimate solution may be, there still exists that appalling number of persons disabled by impaired hearing — estimated as 10 per cent of the total population.

When the loss of hearing is complete, the problem is definitely limited; but residual hearing, whether by air or bone conduction, constitutes a challenge to every otologist. These authors made careful audiometric studies, both by air and bone conduction, of 487 children in a school for the deaf. All of the children had marked degrees of impairment. They found that a complete lack of hearing is rare. Ninety-five per cent of the children responded to at least one frequency. In the great majority, the loss of hearing is bilateral and

approximately equal in two ears. Sex is not a significant factor. Fifty-four per cent of the children were said to have been born deaf. Deafness developing after birth was attributed mostly to meningitis — 17 per cent of the cases. Meningitis, trauma of the head, measles, otitis media and scarlet fever were given as the causes of deafness for almost half of the children. There is apparently no association between the degree of deafness and the stated causes. A significant number of the children were found to have enough residual hearing to warrant some rational effort toward therapeutic relief.

Newhart[57] notes that audiometric surveys of school children have revealed important facts. Lack of progress in scholastic work occurs at least twice as often among children with a hearing defect; speech defects occur eight times more than in normal hearing children; and many behavior problems are often traceable to neglected or uncompensated hearing defects. He considers that children with severe loss of hearing or marked speech disturbances, who cannot continue with the regular classes, should receive instruction in lip-reading, but that they should not be segregated with the totally deaf. Suitable electrical hearing devices should be provided for those who can benefit from their use.

The practical question among school children, according to Kerridge,[58] is, "How deaf are the deaf?" If a child can hear conversation 20 feet away from the teacher, he can continue in the ordinary school. If he can hear less than 20 feet away, but more than 2 feet, he is suitable for the school for the partially deaf. If he cannot hear as far as two feet, he should be educated in a school for the deaf. Of 500,000, 500 children receive special training. This investigation provided the opportunity to compare different methods of testing the hearing — the audiometer, calibrated tuning forks, the human voice and a gramophone audiometer.

The objects of this work are: *1.* To study minor defects of the partially deaf; *2.* to teach speech and vocabulary to the severely deaf; and *3.* to preserve the speech and to enlarge the vocabulary of those children who have become suddenly deaf after the normal acquisition of speech. A. G. Wells, otologist, studied the benefit from the use of amplifiers of speech. The desired benefit was, particularly, improvement of the children's own speech, not necessarily their understanding of the

speech of others. The result — estimated: Three-quarters of the deaf children in the schools would benefit from the use of sound magnification apparatus.

G. P. Crowden[59] reports that the main cause of rejection of men for the British Army has been middle ear disease. Such men are unfit for service, for experience has shown that they are liable to break down under the strain of training — a strain which every young man should not only be able to stand, but to profit by. The average percentage of rejection because of middle ear disease, has been over 4 per cent, and in some years over 5 per cent. In addition, 0.5 per cent who had passed the medical officer in the first instance were discharged for middle ear disease within six months after enlistment; in fact, middle ear disease is the major cause of subsequent discharge from the army during training. Consequently, middle ear disease and other pathologic conditions associated with deafness are major causes of disability in the nation; and any tests or means leading to the detection of such conditions at stages when there is a reasonable chance of cure has a distinct bearing on national health.

By taking "samples" of various sections of the population, and applying the percentage to the entire population of England and Wales, Wells[60] estimates that about 6,500,000 persons, or 16 per cent of the population, have defective hearing in one or both ears — approximately one in every six persons.

In 11 patients with a clinical diagnosis of otosclerosis, Suggit[61] made audiometric studies of the effect on the hearing of introducing normal saline solution into the tympanic cavity through the Eustachian tube. Three cases showed an improvement in hearing. He also tested 15 similar cases who had had no treatment. The audiometric measurements extended over a period varying up to 22 months. Those who had no treatment showed from time to time a variation in hearing acuity comparable to those he had treated by Eustachian injection. Critical examination, therefore, failed to place the improvement of hearing, noted in the three cases, outside the limits of normal variation; and he concluded that no treatment of otosclerosis can be considered of value unless it can produce a sustained improvement in hearing, appreciably greater than 10 decibels over the greater part of the range of hearing.

Guild[62] reminds us that impaired hearing has always been recognized as one of the common afflictions of the aged. That a decline in acuity of hearing with ageing should be regarded as normal has been stated in medical works for the past two or three centuries; and also that the impairment is greater for sounds of high pitch than those of low pitch. So long as tests of hearing were made only with the whisper, the conversational voice, tuning forks, watches, acumeters and whistles, it was impossible to determine the degree of impairment for tones of different pitch. Since the development of the audiometer, it has become possible to make more accurate studies; however, even yet the line between normal and more than normal impairment for any age cannot be sharply drawn. The distinction would be easy if the normal could be defined as best for each age; however, we do know that the average acuity of hearing for high tones decreases with each decade of life. The higher the tone the more is the average impairment. Hearing for extremely high tones is as a rule lost entirely. The majority of elderly people have nearly as good hearing for tones below *high c* as do younger people. Therefore, more than a slight impairment of hearing for low tones, by air conduction, cannot be regarded as the normal condition. By bone conduction of the sound, however, marked impairment occurs so often in old people that it should be regarded as normal. The line cannot be sharply drawn between impaired hearing due to ageing alone and that due to ageing plus other factors. The clinical picture is that of a slight to moderate degree of so-called "nerve deafness"— and this type of impaired hearing is not limited to old age.

Kelley[63] made audiometric measurements in a soundproof room of 168 ears — in subjects who had no history or evidence of ear lesion. He also studied the effect of increasing age on the acuity of hearing music and speech. The tests showed that the hearing for frequencies up to 1,024 cycles remained perfectly normal with increasing age up to 70 years. Between 70 and 80 years, the loss is 14 decibels. At 50 years, presbycusis is already apparent at 2,048 cycles and beyond. The higher the frequency the greater the auditory loss. The elimination of all frequencies above 4,000 cycles does not affect the quality of a violin tone when heard by a person over 60 years. The recognition of vowels after age 60 is not seriously handicapped; however, the recognition of consonants

is inferior. At the intensity preferred by a person with normal hearing for ordinary conversation (38 decibels), the presbycusic person recognizes 75 per cent of the consonants.

Audiometric studies of 100 patients who had auditory hallucinations were made by E. V. Semrad.[64] Of these 100 cases, there were 31 that showed impaired hearing which were classified, "19 toxic deafness, 10 catarrhal deafness, and two nerve deafness." He mentioned that we would have no reason to expect that 19 individuals out of 100, picked at random, would show a "toxic" deafness. Seventeen of these 19 gave a history of excessive alcoholism. In the series of 100, there was no evidence of auditory hyperesthesia. The auditory hallucinations in this group of cases suffering from a toxic type of deafness were essentially of the same nature as those seen in alcoholic psychosis without deafness. Some of the cases experienced hallucinations only in one ear. It was noted that the occurrence of an auditory hallucination during the audiometer examination interfered with the responses of the patient.

Ersner[65] considers that each otolaryngologist should make his own precision measurements, both for diagnosis and the prescribing of hearing aids. By neglecting to do this, we have exposed our patients to a "treatment" by a salesman whose medical scientific background is practically negligible. We cannot deny that there are many skilled and conscientious technicians, some of whom realize their limitations; but the facts irrefutably indicate that the proper person to determine whether or not a hearing device is necessary, and, if so, what its nature should be, is the otologist. Ersner recommends legislative action which would make it unlawful for any layman to sell a hearing aid of any type, except on a prescription and specifications provided by a physician.

This subject is a timely one. This becomes evident when one considers that in the United States today, 15,000,000 people suffer from impaired hearing, about 5,000,000 of whom are deafened enough to require hearing aids. This figure, 5,000,000, constitutes 4 per cent of the population as actual potential consumers of hearing aids. This creates a potential market, even when it excludes all the repairs and replacements, of over $3,000,000,000. For those who are deaf in both ears, a separate instrument for each ear can be worn.

The deaf individual still regards the wearing of a hearing aid as a stigma. Therefore, the deafened as well as the hearing public should be educated to its necessity, just as years ago the public was educated to the use of spectacles.

The object of a hearing aid is to secure better hearing for the individual, prevent mental strain, improve the general well being of the patient and help him mix with normal people; and last, but not least, help improve his social and economic status.

Newhart and Hartig[66] emphasize that the many recent improvements in hearing aids, released with confusing frequency by the competing manufacturers, have imposed on the otolaryngologist certain new responsibilities and problems. His handicapped patients look to him to prescribe or give specific help in the selection of hearing devices best adapted to their individual requirements. If he is to render this service satisfactorily, he must have a certain amount of elementary knowledge of the construction, performance and limitations of these instruments. He must, for example, realize that all carbon microphones give amplification over a very limited range, whereas the vacuum tube hearing aids, with crystal or condenser microphone, when properly designed are comparatively free from distortion. Information afforded by an audiogram for both air and bone conduction is invaluable. It is possible, however, to secure this information with less accuracy by time-consuming tuning fork tests. The audiogram makes clear whether the patient's needs can be satisfactorily met by a carbon microphone instrument, or if he requires a vacuum tube aid, with its greater amplification and fidelity. Until lately, the average person could not tolerate a carbon device unless his hearing loss exceeded 30 to 40 decibels. Today, many of the improved carbon instruments and the wearable vacuum tube aids yield such excellent service that many persons whose loss is only 25 to 30 decibels use them with great comfort and satisfaction. It should be noted also by physicians and educators that many young children with a hearing loss so great that it prevents the acquisition of normal speech, can now learn to talk like normally hearing children by using a modern hearing aid at home and in school.

A comprehensive survey of the subject of deafness appears in a book by Ewing and Ewing.[67] It not only covers a wide

range on the whole subject of deafness but includes a plea for precision measurements, especially for the early detection of a hearing defect, and tests of the efficiency of hearing aids. In addition to an historic survey of audiometry, this book also includes the opinions of many investigators in England and the United States. Also, it will no doubt be read by the hard-of-hearing patients; and it will tend to cause them to look to the otolaryngologist for precision studies and advice.

The authors made hearing tests of many deaf patients who have used hearing aids of the most powerful kind for several years. The majority showed no change in hearing acuity. Some, chiefly those deaf from birth, showed better hearing; this could be explained, in that those who had so rarely heard sound of any kind had not developed sufficient interest to become a skilled and careful listener; and it would appear that the improvement was cerebral. Experimental evidence indicates that the use of hearing aids, far from increasing deafness, sometimes leads to greater skill in the detection and recognition of sound. There is always the patient whose deafness is progressive — who hears less and less, whether he uses an aid or not. Even for such patients, the best course is to enjoy the use of a hearing aid as long as they can. Tests of fatigue are recorded. Those with normal hearing and also those with impaired hearing were exposed to sounds of an intensity up to 110 decibels. *Conclusion:* Pure tones diminished the acuity of normal listeners as much as 30 decibels, but this was always temporary. One deaf patient showed auditory fatigue, but only to a slight degree. No normal or deaf listener suffered any decrease of hearing after listening to very loud speech — due no doubt to the rapid changes in pitch and loudness that occur in speech. The duration of the peak or loudest sounds in speech is never greater than half a second. A few elderly patients found they were unable to benefit from an aid after a few minutes of use — although at first they seemed to be helped. This experience, however, carries its own preventive — the patient discards the aid.

In their comprehensive and significant book on the entire subject of the psychology and physiology of hearing, Stevens and Davis[68] discuss the audiogram. If a person is unable to hear a tone, even when its intensity is so great that his threshold of feeling is reached (the painful upper limit of loudness),

for all practical purposes his hearing loss is 100 per cent. If he hears the tone when its intensity is raised to a value midway between the normal minimal threshold and the upper threshold of feeling, we say that the hearing loss is 50 per cent. It must be borne in mind, however, that this method of designating hearing loss is arbitrary. Care is needed in its interpretation, although this method is convenient and standard. The audiogram is arranged in a logarithmic scale, in decibels. Hence, to say that a person has 50 per cent hearing does not mean that the energy must be raised from zero to 50 per cent of its value at the threshold of feeling in order for him to hear the tone. (Actually, the threshold energy for hearing of a person with a 50 per cent hearing loss is equal to the square root of the energy at the threshold of feeling.)

Again, *loudness* (which is subjective) is not proportional to the logarithm of the stimulus. For example, a tone of 1,000 cycles, plotted on the audiogram as 50 per cent, has a loudness for the normal listener which is only about 1 per cent of the loudness at the threshold of feeling.

Clearly, then, the percentage scale used for recording hearing loss agrees neither with the physical measure of the stimulus nor with the subjective measure of the loudness of the tone. We should bear this in mind; however, the percentage scale is convenient; it has all the advantages of the decibel scale, and its widespread use by those in clinical practice warrants its retention.

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WINNER OF THE 1939 MISSISSIPPI VALLEY MEDICAL SOCIETY CONTEST ANNOUNCED.

The second annual essay contest of the Mississippi Valley Medical Society, "for the best unpublished essay on a subject of practical and applicable value to the general practitioner of medicine," has been concluded. The Annual Awards Committee of the Society has announced that Frederick F. Boyce, A.B., M.D., F.A.C.S., of New Orleans, Assistant Professor of Surgery Louisiana State University, is the winner in a closely contested contest to which many excellent essays were submitted. The winner receives a $100 cash prize, a gold medal, a certificate of award and an invitation to present his essay before the annual meeting of the Mississippi Valley Medical Society. Dr. Boyce will address the Society on the subject of his winning essay, "Toxic Thyroid Disease as a Surgeon Would Have the General Practitioner Conceive It, with a Special Note on the Liver Factor," at Burlington, Iowa, on Sept. 27, 28 and 29. His paper will be published in the January issue of the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). The winner last year was Dr. I. C. Brill, of Portland, Ore., Assistant Professor of Medicine, University of Oregon Medical School, for his essay, "Failure of the Circulation: Types and Treatment," which appeared in last January's issue of the Society's official publication. Because of the nationwide interest in the essay contest, it will be repeated again next year, but plans for the 1940 contest will not be available until November.

STUDIES OF THE WALTZING GUINEA PIG.*†

DR. M. H. LURIE, Boston.

In 1933, Prof. Ibsen, of Kansas Agricultural College, sent us a special breed of guinea pig, called the waltzing or circling guinea pig. This animal is similar to the Japanese dancing (waltzing) mouse. The animal tends to run in circles, either right or left. It has a fine tremor of the head and it is deaf.

All of these characteristics are inherited. The inheritance follows the Mendelian law for a recessive characteristic. There is also evidence of a semilethal effect in the waltzers. The evidence for this is the difficulty in breeding waltzers *inter se*. To keep this strain alive it has been necessary to breed waltzers to normals. The hybrid offspring of such matings are normal in all their reactions, and the ears are normal by histological examination, but when the hybrids are interbred, a certain number of waltzers are produced again. The mortality of these animals is high; a large number seem able to survive only for a few weeks to two or three months.

The animals circle immediately after birth and grow very slowly. The circling occurs as a rule only when the animal is excited or hungry. It may be a large circle with slow movement of the animal, or a small circle in which the animal appears to be chasing itself as fast as possible. They will often circle around another animal. The direction of the circling depends on the individual animal, and is usually in one direction only. The animals are able to run in a straight line for short distances.

The righting reflexes of these animals are normal. The eyes do not respond to stimulation from the vestibular apparatus when the head is moved from side to side, whereas a definite nystagmus is observed in normal animals. The circling of the animals is not affected by enucleation of the eyeballs. Histological examination of the eyeball was normal.

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*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 23, 1939.

†Department of Otology and Laryngology, Harvard Medical School.

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The middle ears are normal histologically and in gross appearance. There is a small incidence of middle ear infection, but not greater than that found in normal guinea pigs.

Studies of the vestibular portion of the internal ear showed normal histological structures throughout. The semicircular canals, their cristae and nerve supply were normal. The utricle and macula of the utricle were normal. The saccule and its macula were also found to be normal. Scarpa's ganglion and the vestibular nerve appeared normal. There was no evidence of abnormal pressure from excessive fluid or collapse of the saccule and utricle from lack of fluid. There were a small number of ears in which there was collapse of the utricle, saccule and semicircular canals. The proportion of these animals was no greater than that found in normal guinea pig ears. This collapse in the few of the ears studied was probably the result of fixation and not a true condition of the living animal. No evidence has been obtained from these animals that the saccule is concerned with the reception of sound.

These findings of a normal vestibular apparatus, normal righting reflexes, a lack of nystagmus from vestibular stimulation (by rotation), indicate that the circling is not due to a peripheral lesion but to a central nervous one. The lack of the eye movements with vestibular stimulation indicated that the lesion might be in the mesencephalon at the region of the red nucleus. Circling has been experimentally induced in normal guinea pigs, and will be reported in another article.

These animals are deaf and the deafness is of an inherited type. The cochlea was not only studied histologically but also by the Wever and Bray method. The animals did not respond to any external sound with a pinna reflex and gave no indication of hearing sounds made near them. The majority of the adult animals studied gave no response to Wever and Bray tests, except a few who responded to low tones up to 300 d.v. at high intensities. Studies by the Wever and Bray method on animals younger than three weeks are now being carried out, for it has only recently been found that the changes occurring in the cochlea begin near term or just after the birth of the animal.

In all animals studied, Reissner's membrane was found to be normal and in normal relationship to the basilar mem-

Fig. 1. Waltzing guinea pig. Utricle and semicircular canal. Normal utricle with normal macula. Normal semicircular canal ampullated end with normal crista present.

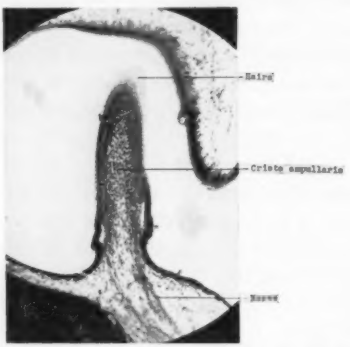

Fig. 2. Waltzing guinea pig. Ampullated end of semicircular canal. Section is at a tangent to show nerve and the hair cells of the crista ampullaris. Normal structures throughout.

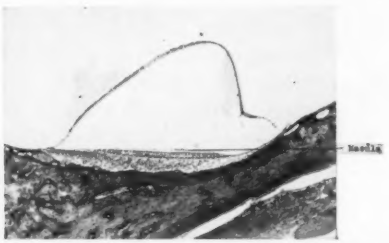

Fig. 3. Waltzing guinea pig. Saccule. Normal saccule with normal structures throughout.

brane. Slight variation of the position of Reissner's membrane was no different than that found in normal animals.

The tectorial membrane is present throughout the cochlea. It is normal in appearance and structure, and its relationship to the basilar membrane is normal. In none of the animals so far examined has it been absent.

The stria vascularis varied considerably in appearance. It was either atrophied or appeared normal in different parts of the same cochlea. In a number of the cochlea there was definite engorgement of the stria vessels. The blood supply appeared normal. No accurate estimate could be made of the relationship of changes found in the stria vascularis to the changes found in the organ of Corti.

The basilar membrane and the spiral ligament showed no variation from the normal.

The organ of Corti showed interesting changes. These changes, when first studied, were thought to be the result of congenital development. Attempts were made to breed animals with definite changes in the organ of Corti. This was found to be impossible. Studies of newborn and intrauterine waltzing guinea pigs showed that the lesions found were not of a congenital type, due to a lack of development, but were of a degenerative type. The evidence at present indicates that this degeneration begins near full term or at birth.

The degeneration of the organ of Corti starts in the first and second turns, and progresses toward the helicotrema. It is not a gradual, progressive degeneration but an irregular one. A great number of the cochlea examined showed a small remnant of organ of Corti nearly normal at the helicotrema.

The degeneration seems to start first in the external hair cells. The internal hair cells degenerate next. The pillar cells degenerate in a similar manner, the external pillar cell disappearing before the internal pillar cell. The degeneration can also take place as a general shrinking of the organ of Corti with the absorption of the hair cells. The original organ of Corti is finally replaced by a simple cuboidal epithelium.

The cochlear nerve degenerates at a much slower rate, and nerve fibres were found in all the animals studied. The cochlear nerve always appears to have more nerve present than one would expect from the amount of organ of Corti present.

The spiral ganglia cells showed degeneration, but here also the amount of degeneration found was less than that expected with complete degeneration of the organ of Corti. A number

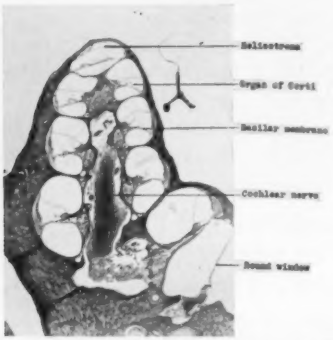

Fig. 4. Waltzing guinea pig. Cochlea as a whole. Guinea pig cochlea has four to five turns. Complete degeneration of basal turns. Partial degeneration of upper turns. Cochlear nerve and spiral ganglion show little, if any, degeneration.

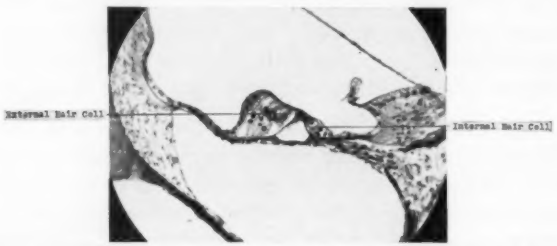

Fig. 5. Waltzing guinea pig. Fourth turn of cochlea near helicotrema. Normal appearing organ of Corti. Internal hair cell shows a cystic formation. External hair cells are present with their nuclei. Note the hairs of the internal and external hair cells.

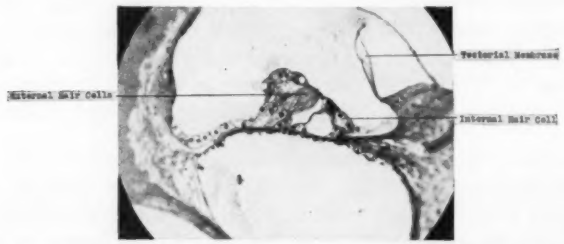

Fig. 6. Waltzing guinea pig. Third turn of cochlea. Degenerating organ of Corti. External hair cells have degenerated. Internal hair cell present and normal in appearance. Note nerve fibres still going through the tunnel.

of the animals one month or less in age showed nerve fibres still going to the basilar membrane, with no organ of Corti present. In all cases studied there was no complete absence of ganglia cells. The older animals have some ganglia cells and nerve present, even with almost complete absence of the organ of Corti. These findings are of great interest, for they seem to indicate that the organ of Corti is not bound up as intimately with its nerve supply as has been thought. Experimental cutting of the VIIIth nerve has shown that the cochlear nerve can degenerate without degeneration of the organ of Corti.

The newborn waltzers and intrauterine waltzing feti at full term were studied. In three animals the organ of Corti was present throughout the cochlea. The spiral ganglia and nerve fibres appeared normal. In the newborn waltzing animal there was some evidence of beginning degeneration or the organ of Corti in the first and second turns. The vestibular portion of the ear was found to be normal. The intrauterine waltzing animals gave no evidence of any changes in the cochlea. From this, we can only conclude that the organ of Corti in these animals develops to a normal end-organ and then degenerates. This degeneration must be due to a gene factor inherited by the animal. As noted before, there has been no arrest in the development of the organ of Corti.

The conception of human deafness must be revised in view of the evidence, from these animals, that deafness can also be due to an inherited degenerative factor. All inherited deafness cannot be designated as otosclerosis; the study of these animals leads to the conclusion that in the human a similar type of inherited degenerative deafness may be present. This may explain the inherited deafness beginning in childhood, at puberty or during adult life, which from its inception does not give the picture of true otosclerosis.

CONCLUSIONS.

1. The circling and deafness of the waltzing guinea pig is an inherited characteristic of the recessive type.

2. The vestibular mechanism is normal and is not affected by the changes in the cochlea.

3. The circling is not caused by vestibular involvement but by disarrangement of a central nervous mechanism, probably in the region of the red nucleus.

4. The saccule does not degenerate with the cochlea. No evidence has been obtained from these animals that the saccule is concerned with the reception of sound.

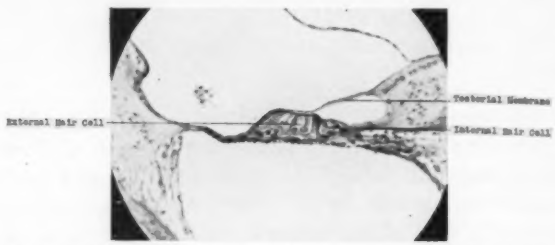

Fig. 7. Waltzing guinea pig. Third turn of cochlea. Organ of Corti degenerating. A general absorption of the cells. External hair cells have completely degenerated. Internal hair cells still present. Pillar cells have become partially absorbed. Tectorial membrane in its normal relationship to the degenerating organ of Corti.

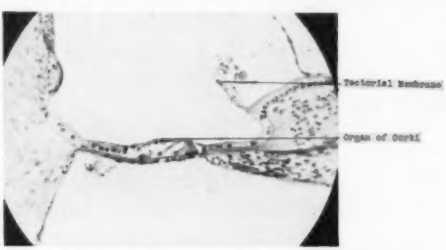

Fig. 8. Waltzing guinea pig. Organ of Corti. Second turn. Showing complete degeneration of the internal and external hair cells and marked absorption of pillar cells and supporting cells.

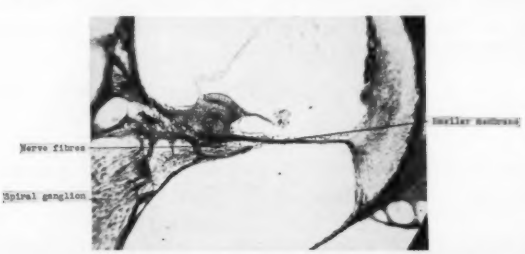

Fig. 9. Waltzing guinea pig. Second turn. Bodian nerve stain. Showing nerve fibres going to basilar membrane with no organ of Corti present.

5. The degeneration of the organ of Corti is an inherited characteristic.

6. The degeneration starts in the organ of Corti and then involves the cochlear nerve. This degeneration begins at birth or near the termination of intrauterine life.*

*These studies are based on the examination of 50 waltzing guinea pigs. The photomicrogaphs by Dr. Werner Mueller. The histological preparations by Mrs. Vivian Hawkins.

483 Beacon Street.

EXPERIMENTALLY INDUCED CIRCLING (WALTZING) IN THE GUINEA PIG.*†

DR. M. H. LURIE and DR. E. W. DEMPSEY, Boston.

For the past five years our studies of the waltzing guinea pig have led us to the conclusion that the circling was due not to a vestibular disturbance but to a lesion of the central nervous system.

Cutting of the VIIIth nerve, or destruction of the labyrinth on one side, did not produce the characteristic waltzing phenomena. The reaction of animals with the VIIIth nerve cut was merely typical of vestibular and cerebellar disturbance.

The conclusion reached by the study of the waltzing guinea pigs was that the lesion might be near the red nucleus of the mesencephalon. The following facts led us to this belief:

1. These animals showed no nystagmus on circling or on movement of the head. The normal guinea pig, on movement of the head, has typical nystagmus.

2. Enucleation of eyeballs of the waltzing guinea pig did not stop the waltzing. The eyes were normal on histological examination.

3. Righting reflexes of the waltzing guinea pig were normal.

4. The vestibular labyrinth proved to be normal in our histological studies.

5. The vestibular nerve and its endings in the medulla were found to be normal.

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*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 23, 1939.

†Departments of Otology and Laryngology, and Physiology, Harvard Medical School.

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The lack of eye movements on vestibular stimulation led us to believe that the lesion might be a break in the reflex arc between the eye muscles and the vestibular apparatus in the region of the IIIrd nerve nuclei in the mesencephalon. This is the region of the red nucleus and the substantia nigra which is thought to have control over posture.

One of us (E. W. D.) in operations on the mammillary bodies of the guinea pig found that a few animals circled when they had recovered from anesthesia. They survived the operation about 24 hours. Study of the brains showed that the lesion was just caudal to the mammillary bodies and involved the interpeduncular nuclei near the exit of the IIIrd nerve from the brain. These lesions were quite extensive and involved most of the midline structures up to and including the dorsal thalamus; moreover, circling did not occur in two animals in which the lesions produced were in the mammillary bodies and other midline structures, but in which the interpeduncular nuclei were not involved. It appeared, therefore, that in order to produce circling, lesions must be placed in the region just caudal to the mammillary bodies.

In operating to reproduce the lesion that caused circling, the mortality was found to be extremely high. Of 30 operated animals, three have survived for more than a month, and five lived only long enough to recover from anesthesia and circle, but died within 24 hours. The immediate death of the animals was due chiefly to hemorrhage or to removal of too much brain tissue. In the five that lived 24 hours, hemorrhage seemed to be the chief cause of death.

The operative procedure is essentially the same as that used for removal of the pituitary body. The approach used is that described by McPhail and Parkes (1933) for hypophysectomy of the guinea pig and hedgehog. Ether anesthesia is used. The animal is placed on its back and incision is made in the median line of the neck from the mandible to the thorax. The tissues on the right side are then separated by spreading of scissors down to the muscles covering the sphenoid and occipital bones. The superior pharyngeal muscles are carefully pushed aside with care to avoid perforation into the nasopharynx. The muscles covering the sphenoid and occipital bones are then split and pushed aside, exposing the suture of the sphenoid and occipital bones. A trephine opening is made just anterior

to this suture with a No. 16 dental burr. This exposes the pituitary body.

The pituitary body, after it has been exposed, is pushed aside, and the dura is then incised in order to reach the interpeduncular region. This incision must avoid the circle of Willis, for, as mentioned before, hemorrhage is the chief cause of failure in the operation. With hemorrhage controlled, a fine suction tube is introduced and with gentle suction a small portion of the brain caudal to the mammillary bodies is removed. If successful, the animal, when recovering from the effects of the ether anesthesia, will begin to move in circles.

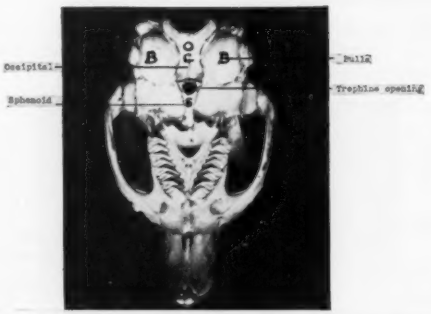

Fig. 1. Guinea pig. Base of skull. Showing site of trephine opening to expose pituitary body. Lesion made in midbrain at about 11:00 o'clock through this opening.

Lesions made in the left side of the brain caused circling to the right (clockwise).

During recovery from anesthesia the animal may be curled up to the right, with the head held slightly up and twisted to the right. The forelegs are pushed in extension to the left. When the animal begins to circle, the motions are rapid, the animal appearing to turn on himself. At times rolling occurs. The pupil of the left eye is in extreme dilatation, and the left eyeball is fixed. The right eye reacts normally to light and vestibular stimulation. There is no evidence of spastic or flaccid paralysis of the fore or hind legs.

With complete recovery from the anesthesia and the operation, the animals react as does a normal guinea pig, except

when excited or disturbed. They then run in circles, and always to the right. There seems to be no attempt by the operated guinea pigs to turn to the left. They can go in a straight line.

In a few days there is further recovery and a beginning compensation for the lesion produced in the animals that have survived. The only evidence at present in two of the three animals living with this lesion are:

1. Dilatation of the pupil of the left eye.
2. Fixation of the left eyeball.
3. The tendency to turn to the right.
4. The tilting of the head to the right.

Fig. 2. Guinea pig. Midbrain section through interpeduncular nucleus. Nissel stain. Showing lesion made on left side. The marked gliosis going to the geniculate body is due to traumatic blood vessel degeneration and is not a degenerated nerve tract.

In the third animal that survived, the lesion produced circling only. There was no dilatation of the pupil, fixation of the eyeball or tilting of the head, but the animal turned to the right.

The similarity of actions by these operated guinea pigs to the waltzing guinea pigs has led us to believe that the cause of the circling in the waltzing guinea pig is a break in the normal reflex arc between the vestibular apparatus, the IIIrd nerve nucleus and the postural centres. The brain of the waltzing guinea pig up to the present time has given us no clue as to this break. The operated animals present evidence

that a lesion in the region of the interpeduncular nuclei may cause symptoms similar to those seen in the waltzing guinea pig.

Further experimentation now being carried out will, we hope, enable us to make a more accurate estimate of the waltzer lesion in the near future.

483 Beacon Street.

DISCUSSION.

DR. STACY R. GUILD (Baltimore): I have had literally only the same opportunity that the rest of you have had to think about this very interesting paper.

My first comment would be on the use of the term "waltzing" to describe these guinea pigs. Those who have seen waltzing mice, from which the term has been adopted, realize that the description of the behavior given by Dr. Lurie scarcely warrants the use of the same term that is used to describe the mice. I hope Dr. Lurie, in the closing discussion, will comment as to the correctness of that assumption on my part. The description reminds me more of the behavior of the so-called toy pigeons, tumblers, rollers, fantails and so forth, all of which forms can walk in a straight line or eat or do other things normally at times, but undergo forced movements at other times under certain circumstances.

I do not question Dr. Lurie's statement that the essential lesion in his experiments is in the interpeduncular nucleus, but the lesions shown in his illustrations do involve other structures. The sections of the ears are beautiful preparations, so there is no question that the peripheral vestibular apparatus is normal. Can it be that the neck reflexes are the cause? One can take, for instance, a pigeon and fasten its head back — I have not done it personally but have seen it done by others — and get most peculiar forced movements.

With respect to the cochlear degenerations, those are most interesting, and I would agree it is degeneration rather than malformation. One point, however, you did not bring out, Dr. Lurie. In looking at the sections as projected, it struck me there was concomitant degeneration of the cells of the external sulcus in those areas in which there was end-organ atrophy. That occurs in man in areas of degeneration of the organ of Corti and seems to precede it. This is described in the paper on The Pathology of High Tone Deafness, by Dr. Crowe, Dr. Polvogt and myself. The relationship I do not know; it may be nutritional.

From all such studies one hopes to be able to get a clue as to the cause of clinical conditions. I feel very doubtful that this investigation gives us any clues as to the Ménière symptom complex. The picture is too different.

I hope, Dr. Lurie, you do not imply that all of us believe the only form of hereditary deafness is otosclerosis. I don't think you meant it thus, but that meaning could be given your statements. The literature, of course, we know is rich in accounts of other forms of hereditary deafness, into which it is not necessary to go at this time.

DR. M. H. LURIE (Boston): "Waltzing" is the term that has been used in genetics to describe this animal. The waltzing mice we have in Boston at the pet shops act exactly the same way. Perhaps my description of a waltzing

guinea pig was not very accurate. I should have brought some moving pictures. At times, these waltzing guinea pigs act exactly the same as the dancing mice do. They simply seem to be a ball as you watch them when they get really excited.

The term I originally used was that of a "circling" animal, but it was objected to because this strain is known as the waltzing strain.

The question of neck reflexes is an interesting one because Prof. Ibsen has written to me saying that he has found a shaker guinea pig. Now, a shaker guinea pig may be the same as a shaker mouse. The head of the shaker mouse is constantly going up and down when he gets excited, and there is a tendency of the animal to fall backwards.

Whether it is the neck reflexes or not that are involved in the lesions we have made in the brain is a question to be solved. Dr. Dempsey has removed structures anterior to this lesion; he has taken out the whole of the mammillary bodies to study temperature changes and has never had one of these animals circle. It is only when the lesion is caudal to the mammillary bodies and in the interpeduncular nucleus that we have had this circling syndrome. In some of these animals we have been able to take out half of the midbrain and the animal has lived, but did not circle at all.

In regard to the statement that we cannot call all deafness otosclerosis, I simply take the viewpoint of what most men doing ear, nose and throat work seem to have taken; that is, that all deafness where the question of inheritance comes in is immediately called otosclerosis. Whether the hearing test shows it or not, the diagnosis made is otosclerosis. I think it is a very important thing to get rid of that misconception, especially as we do find children that will be totally deaf in one ear and have no reason for it, no history of any middle ear infection, and yet not hear anything in one ear. There must be an inherited factor present that causes deafness of this type.

I thank Dr. Guild for this discussion because, after all, he did not read the paper. He has raised important points in regard to some of the interpretations of the facts that we have found.

Dr. William J. McNally (Montreal): May I make a suggestion to Dr. Lurie? He mentioned shaker guinea pigs and his description rather interets me. I would like to make this suggestion because we are able to produce experimentally what he might call shaker frogs by removing the semicircular canals and leaving the utricles intact. On attempting movement or on coming to rest, the animal is greatly disturbed and keeps up a series of what we choose to call pendulations, its head keeps bobbing around and up and down.

The thing that interests me is this: Clinically, one of our main difficulties in interpreting labyrinthine reactions is that we do not know the distribution within the brain stem of the nerves from the individual end-organs. If I understand Dr. Lurie properly, he said that in his waltzing guinea pigs there is an absence of any definite labyrinthine lesion. It is just possible that Dr. Lurie has an animal with a brain stem lesion involving nerve paths from the semicircular canals and that the paths from the utricles are intact. If he can localize that lesion he will be among the first to be able to tell us something about the intracranial or brain stem distribution of the nerves from the end-organs of the semicircular canals on the one hand, and from the utricles on the other.

Dr. M. H. Lurie (Boston): One of the great difficulties in working with animals with inherited tendencies is that you get no evidence of any gross degenerations. Now, those waltzing guinea pigs' brains, as far as we can tell with the different types of nerve stains, cannot be differentiated from the medulla or midbrain of a normal animal. At one time we thought the blood

vessels around the vestibular nuclei were more dilated and numerous than in a normal animal and that this waltzing might be due to the congestion of the blood around the nucleai. But when the slides of normal and waltzing guinea pigs were mixed, our percentage of guesses was so poor that we gave it up.

AMERICAN OTOLOGICAL SOCIETY, INC.

The Seventy-second Annual Meeting of the American Otological Society was held under the presidency of Dr. Friesner, of New York, at the Westchester Country Club, Rye, New York, Monday and Tuesday, May 22 and 23, 1939. There were 80 members, two senior, two honorary and 76 active, and 76 guests in attendance.

The following were elected members of the Council for the ensuing year: Dr. Horace Newhart, Dr. Gordon Berry, Dr. Thomas J. Harris, Dr. Harris P. Mosher, Dr. Isidore Friesner, Dr. Frederick T. Hill and Dr. Ernest M. Seydell.

They from their number elected as President, Dr. Horace Newhart; Vice-President, Dr. Gordon Berry, and Secretary-Treasurer, Dr. Thomas J. Harris.

The following candidates for active membership were elected: Dr. Daniel S. Gunning, 145 East 54th street, New York; Dr. Joseph G. Druss, 1235 Park avenue, New York; Dr. Thomas C. Galloway, 636 Church street, Evanston, Ill.; Dr. Westley M. Hunt, 33 East 68th street, New York; Dr. Frank J. Novak, Jr., 30 North Michigan avenue, Chicago; Dr. Otto Mathias Rott, 407 West Riverside avenue, Spokane, and Dr. Henry Lane Williams, Mayo Clinic, Rochester, Minn.

The Society has lost by death during the past year: Dr. Henry P. Cahill, Boston, active member, Jan. 15, 1939; Dr. Beverly R. Kennon, Norfolk, senior member, Dec. 27, 1938; Dr. Henry O. Reik, New York senior member, June 2, 1938; Dr. Ellison L. Ross, Chicago, active member, Dec. 21, 1938; Dr. Harris H. Vail, Cincinnati, active member, April 10, 1939, and Dr. Antonie P. Voislawsky, New York, active member, Feb. 22, 1939.

The membership of the Society is 151: 12 honorary members, 29 senior members and 110 active members.

SULFANILAMIDE AND ROENTGEN RAY THERAPY FOR ACUTE OTITIS MEDIA AND MASTOIDITIS.*†‡

DR. FLETCHER D. WOODWARD, University, Va.

To evaluate any new therapeutic procedure, it is necessary to compare the incidence, complications and results of treatment of a disease before the introduction of such a procedure with the incidence, complications and results of treatment after adequate clinical trial.

A large number of cases should be studied; therefore, it will be necessary for many observers to report their results in detail, so that when added together an accurate evaluation may be possible.

With this objective in view, we made a statistical survey of acute ear infections over a 10-year period, eight years before the introduction of sulfanilamide, and two years after beginning its use. The results have been charted graphically. We are cognizant of the small number of cases represented but hope that, when added to similar studies, this report may be justified.

The old and accepted methods of treatment have been followed in all cases. Sulfanilamide has merely been added to our armamentarium. The dose has been approximately 1 gr. to two pounds of body weight, by mouth, each 24 hours. Occasionally the drug has been administered parenterally. The usual untoward reactions so completely discussed in the literature have been encountered; however, there have been no serious complications. Sulfapyridine has not been used except in a very few instances. We do not feel that the indications for surgical interference should in any way be influenced by the administration of sulfanilamide.

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*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 23, 1939.

†From the Department of Otolaryngology, University of Virginia Hospital.

‡The cases treated by Roentgen therapy were under the care of Dr. George Cooper, of the Department of Radiology, University of Virginia Hospital. The statistical survey was carried out by Dr. Marion K. Humphries, Jr., of the Department of Otolaryngology. The writer is indebted to them for their collaboration in the presentation of this paper.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

It has been our privilege to witness the dramatic cure of serious streptococcic and pneumococcic infections. The literature contains many such similar instances, so there should be no hesitation in rendering unto Domagk full credit and honor for his original work. In this work we are more interested in the broader application of the drug. Our beneficial results

		BACTERIAL PERCENTAGES IN 200 CULTURES							
		STREP	STAPH	PNEUMO	PROTEUS	HOFMANI	DIPHTH		
OMPA		54	24	8	8	4	2		
SURG MAST.		64.5	21	14.5					

Fig. 1. The percentage of the various types of infections found in 200 ear cultures.

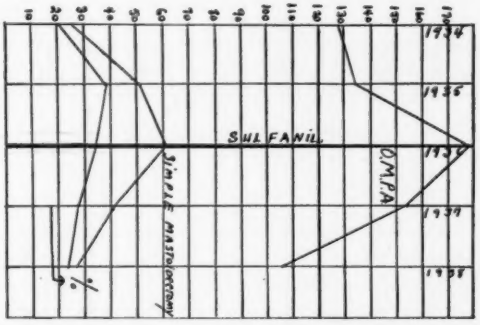

Fig. 2. Seven hundred three cases of acute purulent otitis media over a five-year period, showing a decline in the incidence, the number of mastoidectomies, and the percentage ratio of operation to acute purulent otitis media since the introduction of sulfanilamide two years ago.

have been limited to infections with certain types of pneumococci and hemolytic streptococci, which, fortunately, are the organisms responsible for the majority of acute ear infections.

Staphylococci, S. Viridans, B. Proteus, B. Hofmani, B. Influenza, B. Tuberculosis and Diphtheroids have been the other organisms encountered in our cases. We have not felt that any benefit was obtained in the treatment of any of these by

the use of sulfanilamide. The percentages of the various types of infection found in 200 cultures are shown in Fig. 1.

Horan and French[1] report favorably on the use of sulfanilamide as a routine. In their untreated group there were 607 cases of acute otitis media and 138 mastoid operations, a percentage ratio of 22.7; in their treated group there were

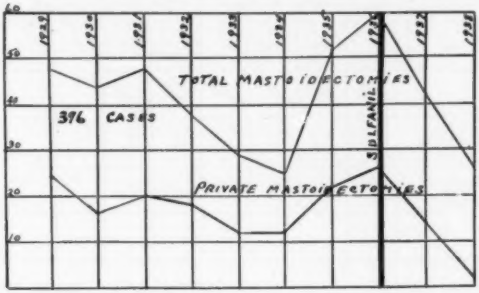

Fig. 3. Three hundred ninety-six mastoidectomies over a 10-year period, showing a decline in both total and particularly in private cases.

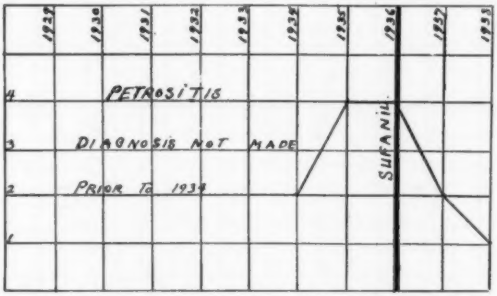

Fig. 4. Petrositis as a complication has only occurred three times in two years.

155 cases of acute otitis media and seven mastoid operations, a percentage ratio of 4.5. The average death rate was 2.85 per cent in the untreated group, and zero in the sulfanilamide treated group. They concluded that the drug prevented complications and that the results justified its use.

Baker and Bradford[2] likewise conclude that the drug is of distinct value in all cases of acute otitis due to hemolytic

streptococci and is probably of value in those due to other organisms.

The accompanying figures show graphically the results of our statistical study.

A decline in the incidence of acute purulent otitis media was noted, with a corresponding decline in the number of

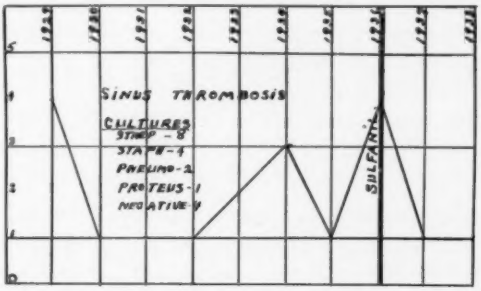

Fig. 5. Lateral sinus thrombosis has occurred only twice in two years.

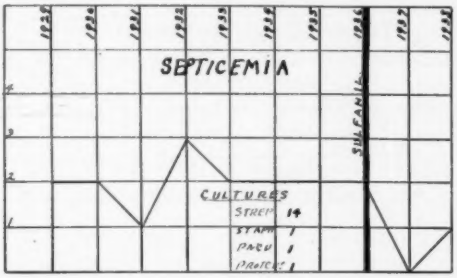

Fig. 6. Septicemia has occurred only once in two years.

mastoidectomies, the percentage ratio of operation to acute otitis media, and in the complications except for meningitis.

Eight cases of meningitis were encountered during the past two years, with six deaths and two recoveries. Although the number of cases is greater here than in previous years, an analysis shows that sulfanilamide could hardly have been expected to prevent their occurrence or to have influenced the result, because the six deaths were due to: *1.* pneumococcus type XIV; *2.* hemolytic streptococcus with epidural abscess

and meningitis; *3.* streptococcus viridans with petrositis and meningitis; *4.* B. influenza meningitis in a child, age 6 months; *5.* hemolytic streptococcus meningitis in a child, age 2 months; and *6.* pneumococus type III with petrositis, epi-

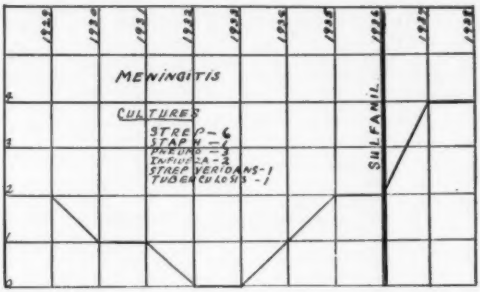

Fig. 7. Meningitis shows an increase, eight cases having occurred in the past two years. Two cases recovered and six died. These are discussed individually in the text.

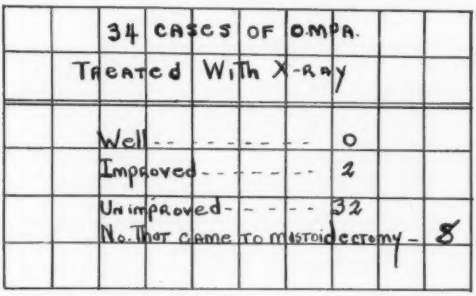

Fig. 8. The results of Roentgen ray therapy in 34 cases of acute purulent otitis media.

dural abscess, cavernous sinus thrombosis, septicemia and meningitis. These conditions were either present on admission, were due to organisms not affected by sulfanilamide, were in patients whose general condition was very poor, or were unusually fulminating types of infection.

There were two recoveries undoubtedly due to sulfanilamide: *1.* pneumococcic type III with otitis media and meningitis;[3] and *2.* hemolytic streptococcic petrositis, epidural abscess and cavernous sinus thrombosis.[4]

Roentgen ray therapy has been widely used for the treatment of infections in various parts of the body, with favorable results in many instances; so to study its effect on acute otitis media, we referred 34 cases to the department of radiology for treatment. They all received adequate therapy, but we were unable to feel that it was of any value in the treatment of acute ear infections, so the study was discontinued. The result is shown in Fig. 8.

Although this report is confined to acute infections, I would like to briefly add that we have noted considerable benefit from Roentgen therapy for reduction of lymphoid tissue in the nasopharynx and about the orifice of the Eustachian tube, for the control of exuberant granulation tissue after radical mastoidectomy, and for polyps and granulation tissue in chronic purulent otitis media.

Conclusions: Whereas the effect of sulfanilamide as shown in these figures seems to have been on the whole favorable, we must consider that this report covers only a small number of cases and is particularly susceptible to seasonal incidence, with which we have all been familiar for a long time.

We agree with the general opinion that it is particularly valuable in hemolytic streptococcic and certain types of pneumococcic infection, and since the majority of acute ear infections are due to these organisms, eventually it will be shown that its routine use does affect the incidence of acute otitis media, the percentage ratio of operations to otitis media, the complications and results of treatment of acute ear infection.

From the small group of cases treated with the Roentgen ray, however, we have not felt that any benefit was obtained.

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DISCUSSION.

DR. JOHN R. PAGE (New York): I regret that no reliable information can yet be gained from the records of the Manhattan Eye, Ear and Throat Hospital in regard to the comparative results of treatment with and without sulfanilamide in cases of acute otitis media and mastoiditis. I found that, in 1934, when sulfanilamide was *not* being used, there were 938 myringotomies and 369 primary simple mastoidectomies recorded for the year. From this, it may be roughly assumed that of those who were not treated with sulfanilamide, 39 per cent required operation on the mastoid. In 1937, when sulfanilamide *was* being used to a considerable extent, 1,029 myringotomies and 389 primary simple mastoidectomies were recorded for the year, from which it may be roughly assumed that of those who had myringotomies performed, 37 per cent were operated upon for mastoiditis. These results are not sufficiently far apart to argue much in favor of sulfanilamide.

But the proportion of mastoidectomies is so high in both years I think the number of myringotomies must have included more severely involved cases than the ordinary acute otitis medias with early mastoid symptoms, for in the series of 836 cases I collected from Dec. 6, 1933, to Dec. 6, 1934, upon which myringotomies were performed there were but 43 mastoidectomies and one death that were under observation until cured (6.6 per cent), and as far as is known in the whole series of 836 cases (5.1 per cent) as compared with 39 and 37 per cent. In this series that had no treatment with sulfanilamide, the 5.1 per cent compares favorably with Horan and French's report of 4.5 per cent of mastoidectomies in 155 cases that were treated with sulfanilamide, which Dr. Woodward referred to.

In meningitis, undoubtedly the most unexpected cures have been effected with sulfanilamide that would never have been obtained without it.

But with its use in acute otitis media and mastoiditis, we have to be more guarded in our interpretation of the signs we have long regarded as reliable indications of mild involvement of the mastoid, and Dr. Law tells me that the Roentgenologist has also been confused by the action of the drug in the mastoid, and that his interpretation of certain films depends on whether or not the case had sulfanilamide. From the fact that most of us have had cases that apparently resolved under this treatment and later turned out to have badly involved mastoids which might better have been operated upon earlier, the opinion seems to be that the drug is not as efficacious in the mastoid as it is in the tympanum or throat or blood and meninges, and that when the bone of the mastoid is definitely involved, one must be doubly careful not to be deceived by mild signs in the canal and tympanum.

It happens more frequently now than ever before that the tympanum apparently clears up and the mastoid continues to smoulder. After all, the large majority of acute middle ear infections require only an early myringotomy to get them well, and some not even that. So, my inclination is not to use this rather tricky drug, for which many have an idiosyncrasy, except in cases that have an obviously severe infection. I never use it in ambulatory cases and never unless its action and concentration can be checked by frequent blood and urine examinations.

Regarding X-ray therapy in mastoiditis, I was impressed by its effect on the mastoid when, three years ago, I operated on a lady with a chronic discharging ear of long duration, who had had several X-ray treatments of her mastoid six weeks before coming to me. It suffices to say that when I saw her she complained of headache, loss of weight, a rise of temperature to 100° to 100.5° every afternoon and a continued discharge from her ear. The essential points revealed in my examination were a large perforation in the drum membrane, a scant odorless discharge, no evidence of cholesteatoma, a leukocyte count of 13,200, with 69 per cent polys., and nothing but the ear to account for it.

Despite the fact that in childhood, 30 years before, the mastoid had been operated upon, a hard and completely reformed cortex was present. When this was removed, a large antral cavity was revealed which was dry, completely free from granulations and lined with a pale atrophic membrane as thin and bloodless as a spider's web. The ossicles could be seen through a wide open aditus and the attic was entirely free from any sign of granulations or thickened membrane. Dry, cancellous, bloodless bone extended deep into the petrous around the semicircular canals. This was removed until the semicircular canals were well revealed. In the lower part of the mastoid, several large cells were opened that were filled with thin pus. One lying on the lower part of the vertical limb of the sinus had a softened inner wall. When this was removed, the sinus wall itself looked to be infected. Absorption apparently had been going on through this area of sinus wall, because after the operation the blood count returned to normal, and a prompt recovery occurred.

This case seemed to me to show the effect of X-ray treatment on granulations in the attic and antrum, and on the mucoperiosteum of the mastoid cells themselves. It also seemed to show that the X-ray treatment did not stop the infection and formation of pus in the lower part of the mastoid over the vertical limb of the sinus.

Nevertheless, as I understand it, the more rational claim for X-ray therapy in mastoiditis is that it promotes drainage from the tympanum, attic and antrum by its selective action on granulations and swollen membrane, and that it is contraindicated in advanced mastoid involvement. From the findings in this case, I think the conclusion as to its effect on granulations and swollen membrane is well founded. But, in closing, let me add that until the X-ray therapists learn more about clinical otology, and the otologists learn more about X-ray therapy and there is closer co-operation between them, I shall continue to take my chance with a good clinical otologist, if I can find one.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The following resolution was adopted at the final meeting of the American Laryngological Association:

"*Whereas,* It has become evident that in many communities and rural districts, little or no attention has been paid by school authorities to disabilities affecting children, especially those resulting from disorders of vision, hearing and speech; be it

"*Resolved:* That the American Laryngological Association approves and recommends the provision of funds by foundations and other appropriate agencies to carry out adequate programs for the detection of visual, hearing and speech deficiencies which constitute a handicap or a threatened handicap to school children, and for providing, through the co-operation of qualified specialists and of State, county and local medical societies, appropriate medical care for indigent and underprivileged children who may be afflicted or threatened with such handicaps."

THE RADICAL MASTOID OPERATION; USE OF THE TENSOR TYMPANI MUSCLE IN CLOSING THE EUSTACHIAN TUBE.*†

DR. HENRY M. GOODYEAR, Cincinnati.

The radical mastoid operation represents the acme of otological surgery in its painstaking application of technique and meticulous aftercare. A successful end-result means an ear free from the danger of extending suppuration, and a dry ear. The hearing after any complete radical mastoid operation is not usually good, and decreases with the years.

As a preventive measure, it is important that all prolonged subacute discharging ears be cured by a careful and extended simple mastoidectomy, and the lower portion of the mastoid incision be kept open, that applicators may be passed to the mastoid antrum until the middle ear is absolutely dry and healed.

This careful watching of the eardrum should persist after every simple mastoidectomy, and serve as the guide to when drainage from the mastoid antrum through the posterior mastoid incision should be permitted to stop. This point cannot be too strongly emphasized. Failure to maintain an open drainage tract from the antrum to the lower end of the incision until the middle ear is healed and the drum no longer inflamed frequently results in a chronic discharging ear. Let the appearance of the tympanum and not the appearance of the posterior wound be the guide as to the progress of the healing.

Small anterior or posterior attic perforations with continuous or intermittent discharge, often scant, foul and watery, indicates a cholesteatoma, and even at the age of 4 or 5 years demands a modified but not a radical operation for the safety of the patient as to life and hearing.

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†From the Otolaryngological Department, University of Cincinnati Medical College.

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It is surprising how frequently cholesteatomata are found in children age 4 to 10 years, who have small attic perforations. These cases can usually be cured by a modified radical mastoidectomy with the preservation of considerable hearing.

Some time ago I saw a woman, age 36 years, who had a small perforation, 2-3 mm. in diameter, posterior in Shrapnell's membrane. After long and careful treatment, the ear had been dry for two years, when suddenly she developed a temperature of 104°, with a chill accompanied by meningeal symptoms. The perforation was dry but upon opening the mastoid a small cholesteatoma and intradural pus were found.

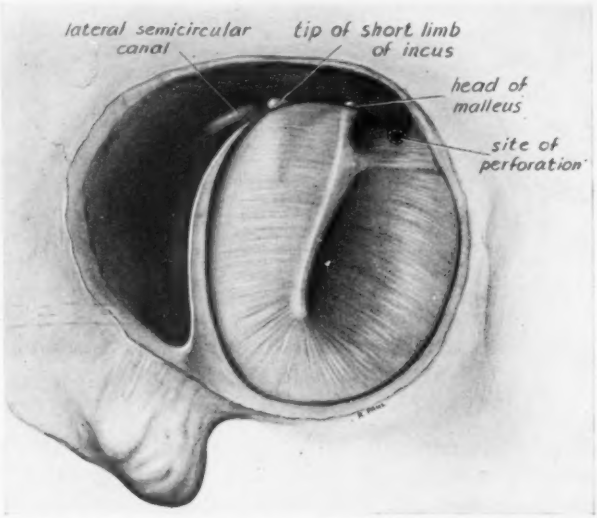

Fig. 1. Modified radical, resected widely in the epitympanic space, extending forward to include perforation.

The patient died. Three months ago a young mother came into the Cincinnati General Hospital, comatose, with an acute exacerbation of an old chronic discharging ear. A brain abscess was present and death left a family of young children without a mother for want of operative relief of a chronic discharging ear.

Central perforations may not be dangerous, but patients with perforations high in the tympanum should never be

released until positively cured, and some sort of surgery will often be necessary to this end.

Temporal and occipital pain occurring with every cold, or acute exacerbation of a chronic discharging ear is a positive indication for a mastoid operation.

For 18 years in all my chronic cases with a small perforation in Shrapnell's membrane I have limited operation to a modified radical (see Fig. 1). The antrum is opened and the posterior canal wall is removed, including the annulus tym-

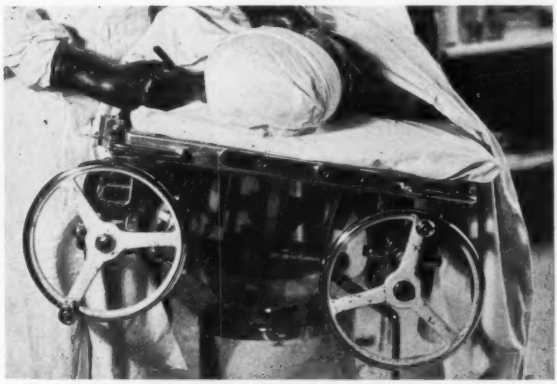

Fig. 2. Table tilted at 25°.

panicus in the region of the aditus ad antrum. Then, with a moderate size Spratt or Richard curette, working parallel with the dura and with the posterior bony margin of the mastoid cavity as a fulcrum, the bone is removed anteriorly, and high in the epitympanic space, until the anterior perforation is reached and made a part of the cavity extending backwards into the mastoid area. The head of the malleus is visible but is not amputated. The outer matrix of the cholesteatoma and the eardrum is disturbed as little as possible.

For the high posterior perforation, a similar procedure has been followed, carrying the dissection forward only to include the perforation in the anterior border of the mastoid cavity. Here, occasionally, it has been necessary to remove the incus.

A circular meatal skin flap, as described by Whiting,[1] is cut, denuded of its cartilage and sutured upward and backward to fascia or periosteum. The cavity is packed with iodoform gauze and the end brought out through the external auditory canal.

The posterior wound, which is curved and cut well into the hairline, is closed with periosteal sutures of No. 0 catgut and the skin is completely closed with silk, usually subcutaneous.

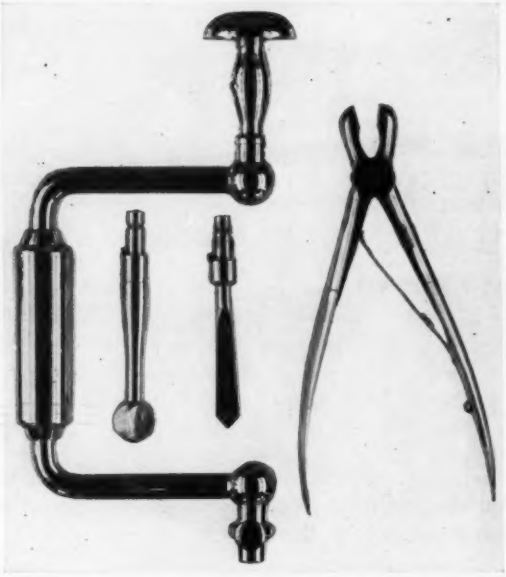

Fig. 3. Hudson perforator and drill. Stelle rongeur.

The iodoform packing is partly or completely removed in five to seven days, or as it becomes soft and wet. Sulzberger powder or boric acid powder is used in healing after carefully drying the cavity, and a 5 or 10 per cent copper sulphate solution is used to keep down excessive granulations.

The radical exenteration of the middle ear is reserved for the cases which show marked destruction of the drum, and ossicles with granulations or polypi.

For the more prolonged and painstaking exenteration and toilet of the middle ear I prefer to tilt the operating table (see Fig. 2) at an angle of 25°, thus easing the working position. Here, too, a high stool is restful in the more tedious drying and completing of the work in the middle ear.

I prefer the Hudson perforator and medium sized burr (see Fig. 3) for the initial opening of the bone in all chronic cases and now use it frequently for opening the cortex in acute cases. The neurosurgeon is constantly more critical of the otologist's use of a gouge and mallet — and should we not open a mastoid in the more delicate manner generally practiced in opening the skull?

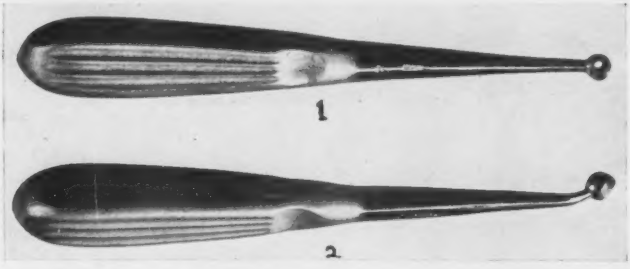

Fig. 4. (1) Round curette, straight shank; (2) Curved shank. (Dr. John D. Richards.)

The production of petechial hemorrhages in the labyrinth or cochlea can, without exaggeration, be conceived with the concussion produced by the mallet.

You will find the use of the perforator and burr easy to master, truly restful and the way of a lazy man in opening sclerotic bone. If dura is exposed by the burr it is not injured, and the outer edge of the burr is directed to lower a goodly portion of the posterior auditory canal; an excellent guide to the antrum.

Once the antrum is exposed, the horizontal semicircular canal is outlined and serves as the most important landmark throughout the remainder of the operation. The posterior cavity is now rounded out, and here a round curette (see Fig. 4) is especially useful, and with the use of the bony edge of the

mastoid cavity as a fulcrum it is a most powerful and rapid cutter of bone.

The bridge is thinned and broken outward by means of a small Spratt curette.

The aditus ad antrum is then widened to the extreme upper limit of the dural plate, and this bone dissection is carried forward, completely eliminating the hypertympanic space.

Fig. 5. Method of shaving down facial ridge (Richard curette).

The facial ridge is lowered by means of a large Richard curette (see Fig. 5). Standing at the head of the table, using the upper edge of the bony cavity as a fulcrum, and shaving downward and outward over the facial ridge, never at any point going below the level of the horizontal canal.

With the ear dry and clean, a Spratt curette is stroked in a similar manner in the middle ear, shaving the posterior canal

wall posteriorly, followed by the use of a small Richards to eliminate the overhang of the hypotympanic space.

By means of a sharp, round curette the anterior wall of the middle ear is shaved down, from within outward to the extreme limit of the glenoid fossae, giving a wide anterior-posterior diameter of the middle ear, and a wide exposure of the Eustachian tube as the apex of a cone extending backward and outward.

The tip of bone over the mouth of the tube is completely removed, and the mouth of the Eustachian tube is curetted

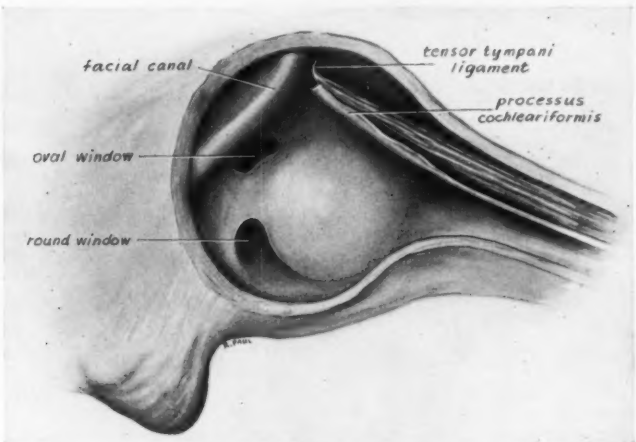

Fig. 6. Tensor tympani ligament; relation to facial nerve.

downward and laterally, and the mucous membrane is removed widely in its upper portion by means of a small Spratt or Barth curette.

I have never removed the mastoid tip in a radical mastoidectomy except where a cerebellar abscess, or lateral sinus thrombosis, was present or suspected. Nor at any time following has it been necessary to remove the tip in any of my uncomplicated cases.

Now comes a step in the operation which I have found most useful and assuring in permanently closing the Eustachian

tube. Unless the tube is closed, intermittent discharge from the ear continues with each cold, often to the discredit of the surgeon and the displeasure of the patient.

After widening the mouth of the Eustachian tube and curetting the tube cells, the posterior end of the tensor tympani muscle is identified by the tensor tympani ligament (see Fig. 6). A small curette, edge medially, is placed under the base of the ligament and moved downward and forward, away from the facial nerve. The muscle is thus elevated from its bed, and the processus cochleariformis is broken down and

Fig. 7. Tensor tympani tucked down into the widened mouth of the Eustachian tube.

removed well into the tube. The muscle is then gently placed or tucked into the widened mouth of the Eustachian tube as a transplant (see Fig. 7).

The origin of the tensor tympani is well down in the cartilaginous part of the Eustachian tube (see Fig. 8); thus, its blood supply and natural attachment is not disturbed. The flap is cut and the cavity packed with 5 per cent iodoform gauze.

I have used this method of transplant in radical mastoidectomies for the past 18 years, not only in my private practice but in rather an extensive clinical service at the Cincin-

nati General Hospital. Even in old chronic ears there is usually sufficient muscle remaining for a transplant, and in many cases I have been surprised by the comparatively large size of this muscle, even in excess of that necessary to close the tube.

Many and varied methods have been attempted in closing the Eustachian tube; among them the following will be mentioned briefly: Pierce[2] used a tongue flap of skin and periosteum from the posterior edge of the mastoid wound, which was drawn into the tube by means of silk drawn through the tube and secured on lead shot. The flap was detached later from the posterior wound. Similar flaps were used by Kahn[3] and Wittmaack.

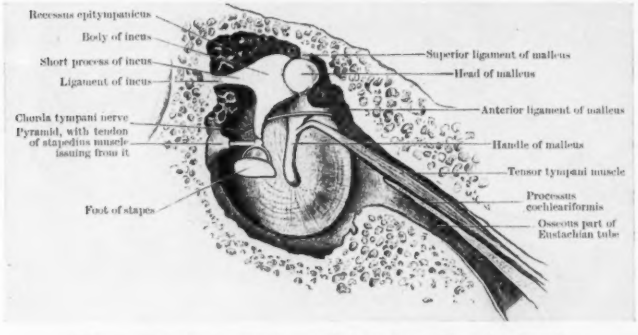

Fig. 8. Tensor tympani, medial surface (Cunningham).

Kirsh[4] closed the tube by tucking in the mucous membrane to form a plug.

Whiting said it had been his custom to apply 1:500 bichloride to the tube, and both he and Dench tried skin grafts over the tube; also tried by Ballance, Gerber, Oppenheimer and many others, who used the Thiersch graft without success.

Heine recommended the folding down of a portion of the tympani membrane, which could not succeed since two epithelial surfaces were in apposition.

Yankauer contrived an instrument for removing the mucoperiosteum of the tympanic part of the tube, which showed

only a small percentage of successes in the hand of McCullagh.[5]

Ferreri used galvanocautery, which endangered the carotid artery.

Holmgren[6] used a mucoperiosteal flap from the promontory, followed by a Thiersch graft to the denuded promontory and the exposed surface of the flap.

Wittmaack and Laurowitsch suggested a wedge of bone or horn.

Olmgren curetted, then laid a piece of periosteum over the opening.

Whitehead recommended the use of a small hand burr in the tube. Allport, the electric drill; while others have recommended various strong caustics and slender electric cautery points.

Passow and Sercer plugged the tube with catgut.

John D. Richards,[7] of New York, recommended the evulsion of the tensor tympani muscle and the breaking down of the cochleariformis in his excellent article on radical mastoidectomy in 1905, and this procedure was taught to me and fellow residents at the Manhattan Eye, Ear and Throat Hospital by Richards, Friesner, Page and others.

I believe, however, that this muscle can be used to an advantage, and I have attempted to preserve it in its entirety to fill the widely excavated upper end of the Eustachian tube as a live muscle transplant, approximate, and naturally attached for its blood supply.

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DISCUSSION.

DR. LE ROY A. SCHALL (Boston): This paper of Dr. Goodyear's is clear, orderly and exact. It is in keeping with his beautiful operating technique. His statement, "let the appearance of the tympanum and not the appearance of the posterior wound be the guide as to the progress of the healing" should be drilled into every house officer.

There is, however, no mention of the diagnosis of cholesteatoma by the X-ray. I quote Dr. MacMillan, Roentgenologist of the Massachusetts Eye, and Ear Infirmary, that by the usual positions of X-raying the mastoids, cholesteatoma can be determined in 45 per cent of the cases. By using the base position, the diagnosis can be made in an additional 25 to 30 per cent, and that in about 25 per cent of the cases it cannot be demonstrated as erosion of bone has not as yet occurred.

I believe there is a place for the endaural operation, and with a careful selection of cases I believe it will become a more popular procedure.

As Dr. Goodyear says, there have been many attempts to close the Eustachian tube. Using the tensor tympani muscle is logical, and should be practical. I am inclined to think that less skillful operators have unknowingly and unintentionally accomplished the same result. I frankly admit that in my radical mastoid surgery, I either do not find sufficient tensor tympani muscle or I fail to recognize it, and that I have not attempted to use it to close the Eustachian tube.

In any discussion as to the causes of aural discharge following radical mastoid surgery, one cannot disregard the words of Almour, "It was thought that many failures to obtain a 'dry ear' were due to the persistent patency of the Eustachian tube. Experience, however, has proven that a large number of dry cavities have patent Eustachian tubes, and that, regardless of how carefully one attempts to close the tympanic orifice, this is achieved but rarely. The proper selection of cases requiring tympanomastoidal surgery has been responsible for the realization that tubal patency is not a prime factor in the failure to obtain a dry cavity. Those chronic otorrheae due to recurring infection of the tympanic mucosa, wherein the nasopharyngeal pathology gains entrance to the middle ear through the tube are no longer submitted to this type of surgery."

THE PREVENTION OF DEAFNESS IN CHILDREN.*†

DR. SAMUEL J. CROWE, Baltimore.

When a child with no evident impairment of hearing for speech, with no tinnitus and no subjective ear symptoms of any kind, is found to have retracted tympanic membranes and an abnormal amount of lymphoid tissue around the pharyngeal orifice of his tubes, the audiometric test will invariably show impairment or loss of hearing for the tones at the upper end of the scale. If the partial tubal obstruction which causes these changes is of short duration and retraction of the tympanic membrane is limited to the pars flaccida, the only tones affected are those far above the speech range. As the function of the tube becomes more impaired and the retraction extends to the anterior quadrant and finally to the entire drum membrane, the hearing for the lower tones begins to be affected. At first the mucous membrane of the middle ear is congested, due to poor aeration, and a few bubbles of mucus may be seen in the ear. Later the hyperemia of the middle ear mucosa disappears and is replaced by a diffuse polypoid or myxomatous condition. At this stage the child has noticeable difficulty in hearing, or is thought to be inattentive or stupid. A series of audiometer tests made on children between the ages of 7 and 12 years show that partial tubal obstruction, never severe enough to cause pain or to direct attention to the ears, first affects the high tones and later causes a progressive loss for tones toward the low end of the scale. This is due to thickening and scar tissue formation in the middle ear, which more and more interferes with the mobility of the ossicular chain.

These symptoms are well known to all of you. In many children they clear up spontaneously or after dietary corrections, removal of tonsils and adenoids, desensitization treatment, or a change of climate. In many others the progress of the disease is so insidious that the middle ear structures have been irreparably damaged before the condition is recognized.

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†From the Otological Research Laboratory, The Johns Hopkins University.

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A normal Eustachian tube is essential for good hearing. Hyperplastic lymphoid tissue in and around the pharyngeal orifices of the tubes is common in children. It may impair the function of the tube and indirectly lead to deafness. Hyperplastic lymphoid tissue in this location can be treated more effectively with irradiation than by either surgery or the usual local measures alone. Paracentesis and aspiration of fluid from the middle ear, the removal of tonsils and adenoids, inflations, and sometimes the application to the tubal mucosa of a mild astringent, may be necessary to supplement the use of radium or X-ray; however, in certain types of conductive deafness in children, irradiation alone has proved to be a most effective method of treatment.

Much adult deafness has begun during childhood and the earlier the conditions causing it are recognized and treated the better the result. Lymphoid tissue is the chief offender.

Except in the relatively acute tubal obstructions or pyogenic infections in the middle ear, the hearing impairment begins with the high tones and may go unnoticed. The progress of the deafness is usually gradual and symptomless until tones used in speech become impaired and changes have occurred in the middle ear which seriously interfere with the movements of the ossicles. We must not assume that by removal of tonsils and adenoids we have preserved the hearing. Indeed, the secondary hypertrophy and hyperplasia of lymphoid tissue in and around the orifice of the Eustachian tubes which often follows this operation in young children may be the direct cause of deafness later in life. Once a condition has been recognized in a child which in any way interferes with the function of the Eustachian tube that child should thereafter be examined by the otologist at regular intervals for the preservation of his hearing, certainly quite as important as the dentist's frequent examinations for the preservation of his teeth.

Lymphoid tissue in the pharynx is more abundant during childhood than after puberty. By tonsillectomy and adenoidectomy we remove only the largest accumulations. In some children lymphoid nodules in the mucosa of the pharynx and Eustachian tubes which cannot be removed hypertrophy after operation and seriously interfere with the aeration of the middle ear. This happens more frequently after operation on young children, and the benefits that may result from removal of enlarged tonsils and adenoids are often temporary so far as the preservation of hearing is concerned.

A Baltimore city health official recently told me that every year the department finds approximately 20,000 public school children who should have their tonsils and adenoids removed, and by using every facility at their command not more than 10 per cent can be cared for. Nothing is done for the other 90 per cent. There are definite indications which make this operation imperative, but in the great majority of children the reduction of hypertrophied adenoids and tonsils twice a year with radium or X-ray would be a very beneficial substitute, particularly if supplemented by the administration of sulfanilamide or sulfapyridine to control the pyogenic complications of the common cold. Children who continue to have frequent colds after a tonsil and adenoid operation, who have lymphoid hyperplasia in the pharynx and nasopharynx, retraction of the tympanic membranes or impaired hearing for high tones must be followed and, if necessary, irradiated two or three times a year until the tendency for lymphoid tissue to regenerate after every cold has disappeared. In addition, they should be examined and treated for allergic manifestations, have the proper food and clothing and, if possible, live in a warm, dry climate during the winter months. These measures will prevent the development of deafness that cannot be cured later in life. Lymphoid tissue in and around the orifice of the tubes is easily infected and not only decreases the lumen of the tube mechanically but stimulates to excessive activity the numerous mucous glands in the cartilaginous end of the tube. Irradiation improves these conditions much more effectively than nasal sprays, nasal drops or the local application of astringents and antiseptics. Irradiation does not destroy glands or permanently remove lymphoid tissue; hence the necessity for repeated treatments to

tide the child over the period during which his hearing is most likely to be damaged.

For diagnosis of this condition in children it is necessary to use a nasopharyngoscope in order to see clearly the pharyngeal orifices of the Eustachian tubes. Neither palpation with the finger nor inspection with a mirror are adequate substitutes. The nasopharyngoscope can usually be passed along the floor of the nose after cocainization, but if a satisfactory examination cannot be made under local anesthesia the child should be given avertin.

Several new points of view must be accepted or proved to our satisfaction before we can recognize and intelligently treat this type of progressive deafness. *1.* Impaired hearing for the higher frequencies with good hearing for low tones does not always indicate an organic and irreparable inner ear or nerve lesion. *2.* A marked shortening or a total loss of hearing by bone conduction for the 512 d.v. fork may arise from a long-continued partial obstruction of the Eustachian tubes, and can often be restored to normal. *3.* Retraction of Shrapnell's membrane in a child, even when the rest of the tympanic membrane looks normal, is a danger signal of impending loss of hearing. It may be due to a partial closure of the Eustachian tube and demands a search for the cause. *4.* Tonsils and adenoids are removed far too frequently in children. This operation may restore the hearing and greatly improve the general health, but in certain children it brings about a condition which imperils the hearing and, we think, is the direct cause of a progressive and disabling deafness later in life. You have all seen the nodules of lymphoid tissue that appear in the pharynx after operation, but unless one uses a nasopharyngoscope it is not so easy to see that the same condition may be present in the nasopharynx and around the orifice of the Eustachian tubes. *5.* If a child's mouth-breathing, recurring ear and throat infections, asthma or some other systemic condition makes it necessary to remove the tonsils and adenoids, we must remember that all lymphoid tissue has not been removed, and what remains may increase in size and extent and be the direct cause of deafness later in life. In order to safeguard the hearing it is advisable to make periodic postoperative examinations of the tympanic membrane and nasopharynx, with special attention to the appear-

ance of the pharyngeal end of the Eustachian tubes and the hearing acuity for the higher frequencies. These examinations should be made once or twice a year until puberty. After this age much of the lymphoid tissue in the upper air passages and Eustachian tubes spontaneously regresses, and what remains does not respond so actively to every infection.

The portal of entry for the pyogenic infections of the upper air passages is lymphoid tissue. The hemolytic streptococcus, the pneumococcus and the staphylococcus are the common infecting organisms. In addition to operative removal of tonsils and adenoids we have two other measures that are of value in combatting recurring infection and mechanical obstruction due to hypertrophied lymphoid tissue, *i.e.*, irradiation with radium or X-ray and the administration of sulfanilamide or sulfapyridine during the early stages of acute infection.

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DISCUSSION.

Dr. Burt R. Shurly (Detroit): The paper of Dr. Crowe appeals to me particularly because I feel that the general field of preventive otolaryngology is a vast field that needs more of our consideration.

For a number of years I have been a member of the Board of Education in the City of Detroit, where we have 260,000 children in the public school system, and I have felt very keenly that the key to the whole problem is the detection of deficient hearing and eyesight among our school children.

Of course, one of the most expensive things in education is to have a lot of children who cannot see and who cannot hear what the teacher is trying to tell them, and it is particularly our work to discover these cases as early as possible, and then there is the problem of how you are going to do this sort of thing and where the funds are coming from.

In Detroit, the Board of Education has put that program on and we have the screening test, where 50 children are tested at a time. We find that in the last 12,500 reported, there was deficient hearing in 4.8 per cent of all the children examined.

Now, in regard to the problem of tonsils and adenoids that we have been working on more or less strenuously for these many years, it seems to me unfair to say that the adenoid operation is not of tremendous value, and the removal of tonsils of the most tremendous value to the growing child if it is properly performed.

It seems to me the adenoid operation is done more poorly and more poorly as these operations are thrown into the hands of novices or internes who learn their job among these children. The adenoid operation is covered up so that the inspection of it afterwards is usually not made, and there are some very badly performed adenoid operations.

The general surgeon and the interne learn to do perhaps very clever, skillful tonsil operations, but their adenoid operations are very bad in many instances.

Again, the LaForce adenotome will not remove all the adenoid tissues, and the great majority of internes and general surgeons doing that work only remove the adenoid tissue in the median line. They leave that important tissue down the pharyngeal wall, and the result is that you have damage that can come through trauma.

We introduced a resolution just the other day in the American Medical Association, asking for their encouragement and help in making a nationwide testing setup for defective school children, and I think this Society ought to encourage that.

I think that is the way to reach that early diagnosis, and it can be done with a comparatively small cost by having a highly trained special nurse and a proper assistant; and the phonograph audiometer, while there is error in it, at least it reaches a large number of children who are deficient in their hearing, and makes them conscious of that, and the parents, also.

We have a system whereby every child in the entire school system has the name and address of his family doctor, and his telephone number, and any deficiency in the periodic examination, including vision and hearing, is referred back to the family doctor.

Now, it is quite well known to us that many family doctors are not particularly interested in problems of hearing or vision, but with the followup system of a splendid corps of nurses, if the condition is not remedied in a reasonable time the followup system reaches the children, and then we have a chance with an early diagnosis to throw that case into the proper channel where it belongs, and to have it come to the otologist or some clinic well provided with the proper examination.

It is, of course, true that we have various kinds of children with tonsils and adenoids. Those very few, nowadays, who are able to pay their way undoubtedly get a nasal pharyngoscope, and a very nice examination, such as Dr. Crowe has described. But in the hurried clinics of our country, they get a rather superficial examination, and the indigent, of whom we have so many, must necessarily be run through these clinics rather rapidly, and many things are left undiscovered.

I believe that the key is the early diagnosis in the school systems of our country. I believe that the problems of nutrition are very important, and when some of our wonderful research men, of whom we have so many in this audience, will tell me and the rest of us why this lymphoid tissue develops as it does, I will be glad to help out, and we will do something great some day.

Dr. Eugene R. Lewis (Los Angeles): This opens up some interesting considerations of the evidence concerning a state of affairs that we have all been more or less involved in, and up against, for a long time.

Of course, the ideal thing to do is to prevent rather than cure. There are difficulties in preventing, however, and prevention has, unfortunately, intangible aspects. When you stop to consider that the more one succeeds in preventing, the less apparent it must be, "intangible" becomes the adjective of choice. It takes an unusual spirit to cast his lot where the more he accomplishes the less it shows, and the more insupportable becomes all proof of accomplishment.

"Prevention" at once sets the scenery one-sidedly; what fails of prevention stands forth so that all may see it, and what actually has been prevented is, de facto, not susceptible of demonstration. Occasionally someone seeks recourse in statistical evaluations of individual data as a means of demonstration, but such demonstrations usually carry little conviction.

I have been long addicted to preventive practices in dealing with clinical problems, and I have thus come to an acute sense of one-sidedness of their settings. Stripped down to the essentials, the facts warrant only quite limited assumptions. Each individual in negotiating the exigencies of living either manages to maintain average levels of hearing, vision and other special sensory functions, or he does not. This at once reduces "prevention" to the undemonstrable level of alternative possibilities.

One's ability to maintain normal special sensory functions is surely enhanced by maintaining intact his mechanismal and sensory equipment for purposes of future service. No matter how much transitory impairment may be experienced in the course of inflammatory, infectious or traumatic disturbance, the possibility of restoring service and winning back to previously normal functional levels must never be disregarded and always kept to the fore among clinical considerations. Within reasonable and safe bounds, recoverability must ever be supreme among clinical considerations, and the goal of rational treatment must ever be the conservation of normal equipment with a view to its subsequent functional value.

Dr. William Mithoefer (Cincinnati): My chief reason for discussing this subject is to emphasize three anatomical factors which have not been mentioned. First, there is no other small area in the body that is as rich in veins and nerves as is the lateral wall of the nasopharynx and the fossa Rossenmüller.

Second, Swoboda and Politzer both demonstrated the fact that the veins of the Eustachian tube communicate on one side with the nasopharynx, and on the other side with the middle ear, so that a slight pressure in the nasopharynx may cause congestion in the middle ear.

Lastly, the mucous membrane of the postnasal space is a very loose connective tissue membrane, which will easily respond to any pressure.

Now, clinically, we all know that in looking at a tympanic membrane in a child, we very often see a slight congestion and an injection of the malleus, and that this congestion and injection along the malleus disappears after the removal of the adenoid.

It has also been demonstrated that if congestion of the tympanic membrane is present, and a solution of cocaine and adrenalin is applied to the postnasal space, the congestion will disappear; therefore, we can deduce that this is a circulatory disturbance, and it is probably for this reason that the high notes are depreciated at this time.

Furthermore, we must remember that a constitutional derangement may be one of the causes. Not only syphilis but also blood sugar shortage, vitamin deficiency, allergy and hypothyroidism must be excluded from the picture.

Dr. Lorente de No (New York): It has been some years since I stopped practicing otology and, therefore, my question may be entirely out of place.

As I understand from Dr. Crowe, the removal of the tonsils and adenoids is not sufficient in a certain number of cases, because there remains a certain lymphoid tissue that has to be removed with irradiation.

Now, this treatment — Is it complicated? Can it be done by an otologist? Or must it be done by a specialist in radiology? And would Dr. Crowe give us some of the details of how to make the irradiation?

DR. SAMUEL J. CROWE (closing): The nasopharynx is irradiated with an applicator small enough to pass with ease along the floor of the nose. Fig. 1 shows the handle, the radon-containing glass tube and the brass tip in which the radon is placed. This brass tip is the screen. One end is closed; the inside bore is 1.5 mm.; the total length is about 1.5 cm.; the outside diameter is 2 mm., and the wall thickness is 0.5 mm. This applicator will hold only 50 mg. of pure radium sulphate, but as much as 1,000 mc. of radon.

DR. T. E. CARMODY: How about the use of radium salt?

DR. CROWE: Radon is used in the applicator because the time required for the treatment is thereby much shortened. Radium salts are so bulky that not more than 50 mgm. can be placed in an applicator that will easily pass through the nose of a child.

A safe dosage for lymphoid tissue in the nasopharynx of a child is 1.5 to 1.8 gm. minutes on each side for the first treatment; a month later, from 2 to 2.5 gm. minutes on each side may be given if necessary. The dosage in gram minutes is calculated by multiplying the number of millicuries in the applicator by the number of minutes the applicator is in the nasopharynx. Thus, if the applicator contains 600 mc., the minimum exposure would be two and one-half minutes, and the maximum four and one-fourth minutes on each side.

DR. HORACE NEWHART (Minneapolis): Before he leaves the platform, may I ask Dr. Crowe one question? In order to avoid a possible misapprehension regarding the effectiveness of audiometers of different types, will you kindly explain the present limitations of the ordinarily used phonograph audiometer in disclosing hearing deficiencies for the higher frequencies as compared with the pure tone audiometer?

DR. CROWE: It is certainly inadequate for the detection of early impairment of hearing. When the tones in the speech range have become involved, the deafness is pretty far advanced. If the child is young, he seems to respond to treatment much better than when he is older. If the child is 16 or 18 years of age, however, no method of treatment seems to help very much.

The real solution of the problem in school children is to detect the changes in hearing long before they can be detected with the phonographic audiometer.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Forty-fifth Annual Meeting of the Society was held at the Drake Hotel in Chicago on June 9, 10 and 11, 1939. There were 137 members and 85 visitors in attendance; a total of 222.

Twenty-five candidates were elected to Active Fellowship. Dr. Chevalier Jackson was elected an Emeritus Fellow. Dr. Jackson has been a member of the Society since 1901, was its President in 1911, and has always been one of its active and most distinguished members.

In addition to the officers listed above, the following Council members were elected for the ensuing year:

Class A: George M. Coates, Philadelphia; John J. Shea, Memphis, and William Mithoefer, Cincinnati.

Class B: Samuel J. Kopetzky, New York; H. Marshall Taylor, Jacksonville, Fla., and Millard F. Arbuckle, St. Louis.

Class C: H. I. Lillie, Rochester, Minn.; James G. Dwyer, New York, and Frank R. Spencer, Boulder, Colo.

The Section Meetings will be held in January, 1940, as follows: Eastern Section, Pittsburgh; Southern Section, Columbia, S. C.; Middle Section, Kansas City, Mo., and Western Section, Los Angeles.

Subject to the approval of the Council, the 1940 Meeting of the Society, under the presidency of Dr. Lee M. Hurd, will be held in New York City early in June; and the 1941 Meeting, under the presidency of Dr. J. Mackenzie Brown, will be held on the Pacific Coast.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of April 19, 1939.

SYMPOSIUM: ACUTE CONTAGIOUS DISEASES—EAR, NOSE AND THROAT COMPLICATIONS.

(Continued from June issue.)

(e) Poliomyelitis. Dr. Philip M. Stimson.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. A. HARRY NEFFSON: I wish to compliment the speakers on their excellent presentations and to thank them for a very interesting and instructive evening. Not having seen these papers beforehand made it all the more enjoyable. When Dr. White invited me to take part in the discussion, he rather optimistically, I think, suggested that I cover all the papers presented. Even if I felt qualified to do this, time would certainly not permit me to do justice to them all. I will therefore confine myself to making a few remarks on some of the papers, and spend the rest of my time on the subject with which I am most familiar, and which has been a pet interest of mine, namely, croup.

Dr. Dolgopol and Dr. Stern have described the mechanism of the production of pneumothorax and mediastinal emphysema very well and have mentioned several reports confirming this view, and yet, in spite of this, whenever a tracheotomy is done and pneumothorax occurs, medical men and particularly pediatricians are wont to ascribe the pneumothorax to the tracheotomy. A greater familiarity with this mechanism, I am sure, will remedy such a situation.

I would like to cite a very graphic illustration of the occurrence of pneumothorax before a tracheotomy had been done. A two-year-old child was admitted to the Willard Parker Hospital in severe respiratory distress, suffering from influenzal laryngotracheobronchitis and supraglottic edema. Immediate tracheotomy was decided upon. While the pretracheal tissues were being separated, an unusual phenomenon was noted—there was a ballooning of thin apical pleura into the wound, covering the trachea. With each expiration this ballooning could be seen, making tracheotomy impossible without puncturing this pleura. The pleura was retracted with difficulty and the tracheotomy done. The explanation of this occurrence was not established until some time later, when an X-ray examination confirmed the diagnosis of pneumothorax. Evidently the air in the pleural cavity was compressed with each expiration and displaced to the point of least resistance, in this case the tracheotomy wound. I mention this instance as a caution to anybody else who may happen to have a similar experience, in order that the presence of a pneumothorax may be suspected.

I don't know whether it was stressed enough tonight that an X-ray should be taken routinely before and after tracheotomy in order to establish the presence of pneumothorax. Physical examination alone is not enough, because very often hyper-resonance is found, and a diagnosis of pulmonary emphysema is made when there is really a pneumothorax. Physical signs are sometimes almost identical in the two conditions and the diagnosis cannot be established

unless an X-ray is taken. When the diagnosis is established, immediate treatment is necessary. The air should be evacuated as quickly and effectively as possible, and this should be repeated as often as is necessary. I think a pneumothorax apparatus should be used, so that the pressure can be ascertained each time and the air removed until the negative pressure approaches normal limits.

Dr. Wilson has mentioned peritonsillar abscess, but evidently did not have enough time to discuss it. I think this subject is worth a few remarks. I have seen six cases of diphtheria in adults admitted to the Willard Parker Hospital after having undergone incision for peritonsillar abscess by otolaryngologists. These incisions were made by private physicians in two cases, and in leading hospitals in four cases. Two patients were in doctors' families. The mortality was appalling — three of the six patients died. Two developed severe myocarditis and had a very protracted convalescence. The reasons for this high mortality and morbidity rate are that the incisions opened up wide avenues for the extension of the disease and also the absorption of the toxin, and diphtheria antitoxin was not administered until four to seven days after the onset of the disease. These mistakes, I think, are made plainly because doctors are wont to consider diphtheria as a disease of childhood and, consequently, when an adult presents himself they fail to consider that diagnosis seriously. I have seen cases of diphtheria as late as four or five days in which there is only edema and redness of the peritonsillar tissues. with very little membrane. In these cases it is very easy to see why a mistake could be made and an incision for peritonsillar abscess be performed. There are certain distinguishing features: In diphtheria there is a low grade fever, proportionately rapid pulse, no history of severe sore throat and chills, the edema is usually bilateral and there is an adenocellulitis of the neck and no local fluctuation. In an abscess, on the other hand, there is usually a history of severe sore throat, high fever, unilateral edema with local fluctuation, and opening the mouth is very difficult. If you attempt to depress the tongue, the pain is excruciating, because the muscles compress the abscess. When in doubt, it is always wise to wait 24 hours and take a culture. If the so-called peritonsillar abscess is opened and no pus is obtained, then the patient should be given antitoxin at once, or a rapid culture should be done. It is possible to get a report as soon as four hours. I have never seen a peritonsillar abscess in diphtheria requiring incision after an experience of over four years as resident at the Willard Parker Hospital; however, it does occur rarely.

(To be continued in a succeeding issue.)

BOOK REVIEWS.

The Personality Structure of Stuttering. By James F. Bender, Ph.D., C.P., Chairman, Department of Speech, Queens College; Director of Information, American Speech Correction; Co-Author of Speech Correction Manual; Principles and Practices of Speech Correction; Speech in College and Life; Phonetic Readings in American Speech. One hundred eighty-nine pages with Index and 14 Tables. New York: Pitman Publishing Co., 2 West 45th street. 1939. Price $2.00.

A profound study in psychology of stuttering. Bender estimates the validity of Bernreuter's "Personality Inventory" more than others and selects it for his own investigations, made on a group of male college stutterers. "They constitute the most readily available, homogenous group of stutterers since there are more speech clinics conducted by experts in the colleges and universities than in the secondary and primary schools in the United States." He describes the conflicts of theories (i.e., the trait and antitrait point of view), the characteristic physique (biochemistry, vasomotor condition, tremor rates, voluntary movements, breathing actions, reflexes, vocal anomalies, eye movements, motor co-ordination, intelligence, silent reading, mirror reading, handedness). The result of the measurement is that the stuttering group was significantly higher neurotically, more introverted, less dominant, less self-confident, less sociable and of avoidance behavior.

Why did the writer not use the great help of an exact observation of the fine clinical symptoms in order to enlighten the stuttering problem?

E. F.

The Handicap of Deafness. By Irene R. Ewing, M.Sc., Ellis Lloyd Jones Reader in the Education of the Deaf, University of Manchester, and Alex. W. G. Ewing, M.A., Ph.D., Honorary Special Lecturer in the Education of the Deaf, University of Manchester. With illustrations. London: Longmans, Green and Co., Ltd. 1938.

The authors deserve much credit for their pioneering work, in "ways of alleviating incurable deafness." They present a comprehensive study of the deafened child, much of which is in nontechnical language. It includes an account of the system used at Manchester University for examining, classifying and teaching children with organic hearing defects, and repeatedly emphasizes the importance of the combination of electrical amplification and lip-reading, the latter being especially stressed in their teaching methods. An appeal is made for early thought on the subject of loss of hearing in order to prevent serious deterioration of speech.

Consideration is given to audiometry and the use and misuse of hearing aids of various types. Unfortunately, the English terminology describing hearing aids does not coincide with our own and is confusing, but important problems regarding amplification, etc., are discussed in detail. Attention is called to such points as age variations in audiometric curves, essential requirements of a reliable audiometer, possibilities of errors in the response of the subject, and a thorough discussion of results of large group-testing. Many audiometric curves are reproduced to explain points under discussion.

Despite the ambitious intentions of the authors to write a book which the laity might understand and appreciate, it has definite limitations in this respect. For the psychologist, the teacher of the deaf and workers in the field, this is a manuscript worthy of serious study and consideration. To the otolaryngologist who is daily confronted with the problem of progressive deafness, and who may be particularly interested in deaf prevention as well as treatment, I urge careful perusal of the many practical points which are too often neglected or entirely omitted in otolaryngologic teaching. B. H. S.